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HEREDITARY OPTIC ATROPHY AS A POSSIBLE MENACE TO THE COMMUNITY.[1]

By C. Morlet, D.S.O., M.B., B.S. (Melb.),
Perth.

I PROPOSE to-night to direct your attention to a very dreadful and, as yet, rare condition of the eye which has forced itself upon my notice since I became a resident of this State.

The disastrous consequences of the disease, as well as its strange ætiology and distribution, should render it of extreme interest to the general practitioner, as well as to the specialist.

I, therefore, will commence with a brief description of the condition itself, so far as we are at present conversant with it.

At the outset, I would emphasize the fact that hereditary optic atrophy, though its importance is acknowledged on account of its dire results, has always been accepted as a rare condition, interesting mainly from a historical point of view, and has been regarded rather in the light of a pathological curiosity.

Fear of the possibility of this disease emerging from its depth of obscurity and appearing in our midst as a very real and serious factor among the problems which daily face the general practitioner, is my reason for calling attention to the following facts.

Symptomatology.

Hereditary optic atrophy was first adequately described by Leber in 1888 and is sometimes referred to as Leber's disease. According to this authority, the disease developes as a retro-bulbar neuritis, usually a few years after puberty, around the twentieth year. The onset is dramatic. A previously healthy and apparently normal individual is suddenly stricken with defective sight. Without any warning, vision becomes misty and dim; colours cannot be distinguished and within two or three weeks the unfortunate patient may have to be led about, so intense has become the visual defect.

For several weeks after the onset, no ophthalmoscopic change whatever may be detected, though sometimes slight indications of optic neuritis, such as blurring of the margins of the disc, etc., become evident quite early. Before very long, however, evidence of commencing atrophy, often at first only in the temporal quadrant, becomes apparent, heralding permanent and incurable destruction of sight.

It is now found that the visual defect has assumed the form of a very large and very intense central scotoma, which blots out all central vision from each eye, while the hazy and shadowy sight, which

[1] Read at a meeting of the Western Australian Branch of the British Medical Association on July 20, 1921.

is derived from the periphery of the retina, is little interfered with. This central scotoma, once developed, is permanent and incurable, whatever improvement of general vision may ultimately take place.

Both eyes may be equally and simultaneously attacked, or one may be affected before the other. In any case, both eyes are ultimately involved.

Treatment of the condition is of no avail.

Prognosis.

Total blindness, according to Leber and also to Nettleship, never ensues. On the contrary, a certain degree of improvement of vision is the rule. Usually this improvement is not great, though often sufficient to permit the patient to get about without assistance. A few cases, however, have been reported in which the patients have got back quite useful vision.

The improvement varies in extent and in time of commencement and may be unequal in the two eyes. The sight may commence to return as long as eighteen months after the onset of the disease.

Ætiology.

(1) Sex.

The condition is essentially hereditary and affects almost exclusively males.

Females are but rarely attacked, but are always instrumental in the transmission of the disease. Hence, the sons of a family inherit the condition by means of the unaffected mother, though it may be unknown in the forbears of the father.

Consanguinity of the parents is very unusual.

(2) Age.

Though usually occurring about the age of puberty or shortly afterwards, the onset may be delayed until later in life and is then commonly in the fourth or fifth decade. Cases have been reported with an onset as early as the first and as late as the sixty-seventh year.

In the rare cases in which females are attacked, the onset is often quite early, before the fourteenth year.

The disease commonly runs the same course, as regards age of onset and prognosis, in the various members of the same family.

(3) Cause.

The condition consists of a selective invasion of the papillo-macular bundle of nerve fibres, of which the cause is quite obscure.

Various explanations have been advanced, one of the more attractive having reference to the development of the sphenoid bone. This bone, in which the optic foramina are situated, does not complete its development until fairly late and it has been suggested that hereditary optic atrophy, with its onset about the age of puberty, may be due to some irregularity in the growth of this bone, producing pressure on the optic nerves.

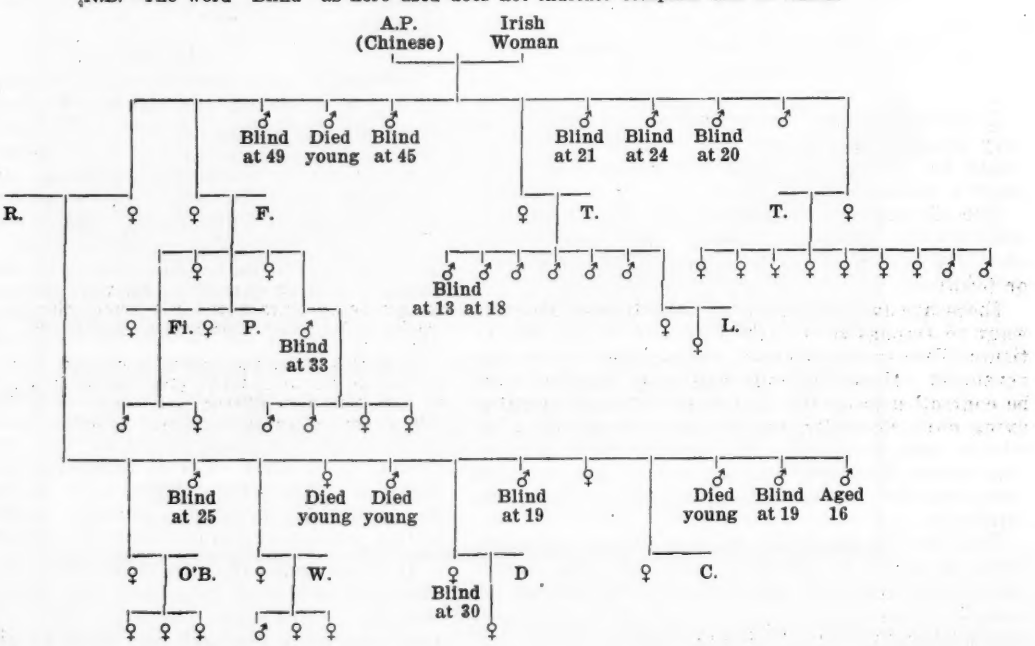
This explanation is unsatisfactory, apart from its vagueness, for many of the reported cases.

Diagnosis.

The condition has to be distinguished from the family optic atrophy due to tower skull or oxy-

GENEALOGICAL TABLE.

N.B.—The word "Blind" as here used does not indicate complete loss of vision.



cephaly and from other cranial defects which involve pressure on the optic nerve.

The entire absence of any obvious explanation for the sudden visual defect, coupled with the family history and the rapid development of the disc changes, usually serves to disclose the identity of hereditary optic atrophy and allows but little risk of confusion with other conditions.

The Affection in Western Australia.

With this brief sketch of what is known of the condition, I submit to you the genealogical tree of a Western Australian family, who resided for many years at Broome.

The various members are now scattered throughout Australasia, branches having settled in New South Wales, Queensland and New Zealand. Wherever they are, these unfortunate people disseminate their family disease broadcast, by the simple process of marrying into other families and reproducing their kind.

I am in touch by correspondence with all the affected branches of the family and have satisfied myself that each member who has lost his sight, does actually suffer from optic atrophy.

As you will see by a glance at the diagram, this is an exceptionally prolific family. You will notice that in the four generations which are recorded there are at present living 22 males and 31 females. Of these 22 males, ten are boys under 19 years and twelve are adults. Of the twelve adults, only one has so far been spared his vision, the remaining eleven being all partially blind men. Eight of them became so about the age of puberty and three in later life, between the ages of 35 and 50. So that the one who has so far escaped, cannot, therefore, be declared safe, but may also become a victim at any moment.

Of the 31 living females, seventeen are still children, while fourteen are adults. Of these fourteen adults, eleven are already married and nine of them have children. Only one female has ever suffered from the disease; she is here to-night.

By the act of marriage, each of these women may introduce into the family of her unsuspecting husband the seeds of her own family complaint and her children will probably grow up as either sufferers from or transmitters of hereditary optic atrophy, depending only on whether they be male or female.

There are two points about this disease which I want to emphasize to-night before showing the patients: Firstly, the disease is incurable once it has developed. Hence the only means by which it may be controlled is by the fact of the affected families dying out. Secondly, the disease is rare, which is to say that comparatively few affected families have been reported up to date.

The family I am reporting to-night, however, is a prolific one. It contains at present twelve individuals who actually are sufferers and ten boys who have not yet attained the age. It contains seventeen young females as yet unmarried, any or all of whom may be instrumental in giving rise to fresh affected families. From the union of A.P. with the Irish woman, twelve entirely distinct families have already accrued.

Considering the sparseness of our population, I think that these figures are, to say the least, alarming.

I believe that twenty years hence the number of cases of hereditary optic atrophy in Australia will have increased out of proportion to the increase in her population and I foresee that this condition might, conceivably, some day appear as a definite menace to the community.

Obviously, the only means of stamping out this menace is by preventing, as far as is practicable, the further proliferation and spread of any affected families. Difficult as this will be to bring about, I think everyone will agree that it is our obvious duty to the community among whom we live and work, to do what we can to this end.

I have brought with me to-night the two latest victims in the family, who both belong to the branch still resident in Western Australia, and have here a brief medical history of each case.

The patients themselves will be glad to answer any of your questions and submit to any examination you may wish to make.

The girl, Mrs. D., is aged 30 years. She is the only female member of the family who has up to date been affected.

She has lately given birth to her first child, an infant daughter, who is now eight months old. This baby, you see, will possibly, twenty years hence, be a transmitter of the family disease, though probably not a sufferer as her mother is.

The boy, S.R., is, fortunately, almost the last of his large family, there being only one younger than he and that a lad of 16. In three years' time, this boy also will be approaching the dangerous age of 19 or 20.

Case Reports.

Mrs. D., aged 30 years, has always had good sight and has never worn glasses. In November, 1920, she gave birth to her first child, a daughter. In January, 1921, her sight began to get dim in the right eye. Within a few weeks the sight of both eyes became very bad indeed and it has been getting rapidly worse.

She was first seen on May 17, 1921. Her vision was then as follows: The right eye could count fingers at a distance of 30 cm.. The left eye could distinguish shadows only. There was a wide, dense, central scotoma before each eye. The pupils were dilated and both reacted very sluggishly to light; both discs were definitely pale and atrophic. The left disc, *i.e.*, the side most recently affected, showed undoubted signs of a subsiding optic neuritis, the margin being blurred and the whole disc being a little swollen. No other pathological change was evident in either eye.

Since this time, the central scotomata have increased, the vision has diminished proportionately and the pupils have become more dilated and less active to light.

Now, both discs are definitely atrophic, there being no sign of active neuritis.

S.R., aged 19 years, single, has always had good sight. He has not worn glasses. He was always backward at school, but otherwise a normal, healthy lad. He was first seen on April 6, 1921. Two weeks previously, whilst at the races, it was found that he had difficulty in distinguishing the colours worn by the jockeys; it was noted that his left eye was more dim than his right. Since then the vision of each eye got worse daily.

When first seen the vision was: Right eye, $^{3}/_{60}$; left eye, shadows only. The visual defect consisted mainly in a

very dense and expansive central scotoma. Physical examination of the discs and fundi generally disclosed no abnormality. The pupils were equal; both reacted to light, but somewhat sluggishly.

In a few weeks the vision became still more defective, central scotoma persisting and increasing in area and in density. The pupils dilated as vision failed and became still less responsive to the light stimulus.

By April 20 optic atrophy was becoming evident to the ophthalmoscope and is now quite obvious.

The boy has a blood pressure of 160 mm. and a somewhat enlarged heart. Otherwise his system organs are normal. There was no response to the Wassermann test.

PLAGUE CONTROL IN OTHER COUNTRIES.[1]

By J. S. C. Elkington, M.D., D.P.H.,
Chief Quarantine Officer, North-Western Division, Australian Health Service.

THE general principles on which bubonic plague has been and is being successfully fought in other countries, may be stated in four short sentences:

(1) Plague is, originally, a disease of rats and certain other rodent animals.
(2) It is transmitted from rat to man by certain species of rat fleas.
(3) Human patients do not play any important part in spreading bubonic plague.
(4) So-called "insanitary conditions" contribute to the spread of plague only so far as they afford food and shelter to rats.

These statements are not guess work or mere ideas. They are based on exact observation and proved by years of practice. The modern fighters of plague obtain their successful results only by the accurate and intelligent application of exact knowledge of the habits and disease possibilities of rats and their fleas.

Pneumonic plague need not be considered here at any length. It is practically uncontrollable when it occurs on any large scale, but the outbreak soon exhausts itself by the death of practically all infected people and the flight of most of the non-infected. It probably needs certain special conditions which, nowadays, are found but rarely in any community possessing any real degree of civilization, before it occurs on any really large scale. Those who study epidemics and their history, realize, however, that there is always a possibility of a form of influenzal plague arising from some unforeseen bacterial combination which might conceivably remove a large proportion of the human race in a very few months.

In 1903, plague appeared in San Francisco. The city and state authorities treated it as a political matter, attempted to conceal it for as long as possible, arrested a Federal health officer for doing his duty and dismissed the state bacteriologist from office because he found plague germs. The public was misled by statements that plague was a mediæval disease which could not do any harm at the present day, that it attacked only Chinese or coloured people, that it was only a scare got up by the Federal health people and that rats were natural scavengers which it would be dangerous, even sacrilegious, to attack. So effective was this campaign in preventing anything from being done that, after 100 deaths or so had occurred, the neighbouring states got together and told the San Francisco politicians that if they interfered any more with the people who understood plague, the city and state would be put under general quarantine by combined self-protective action of their neighbours. This phase is mentioned as an illustration of plague control by political methods and its result.

The next chapter opened under the control of the Federal health officers, who had previously had no actual power to do anything. It lasted eighteen months, included every known means of rat destruction and involved the reconditioning of quite a considerable section of the city, but it got rid of that particular infection of plague.

In 1907 San Francisco was devastated by an earthquake and a fire. On top of this came a fresh infection of plague. Except for the rebuilt parts, which had been plague-infected four years before, the city was a rat paradise. After some fifty cases had occurred, the city decided, after recollecting previous experience, to drop politics and get busy. With the assistance of the Federal Health Service a laboratory was installed, the city was divided into thirteen districts, each with a skilled medical officer in charge, and a staff of inspectors, assistants, foremen and labourers, and the whole operations were controlled from a headquarters office. Before the operations finished the laboratory had examined 154,000 rats, every stable of the 5,292 in the city had a concrete floor, every warehouse, market, bakery, produce store, restaurant, hotel, lodging-house and fowl-yard was rat-proofed, over 1,700 houses had been demolished and over four million square feet of concrete laid in basements and under floors. More than a million inspections of premises were made and 350,000 rats were caught. Ten million poisoned baits were laid. The temporary staff employed numbered some 900 men and not a single one of them was appointed by any other authority than the Federal officer in charge of the work; there were no political appointments.

How was all this done? It was done by the only method which can get the full weight of any community behind a movement, namely, by the citizens themselves. At the outset of operations a "Citizens' Health Committee" was appointed by the mayor; they appointed an executive committee and the executive committee organized sub-committees covering every branch of the industrial, professional, religious and business communities, men and women alike. They subscribed over £35,000, issued 290,000 circulars and countless posters and notices in three languages and gave an inestimable amount of volunteer help. Without them the whole organization, Federal and State, would not have attained success in one-half the time it did, for it was they who educated the public to cease furnishing shelter and food for rats and to regard rats as bitter personal enemies. Before they finished, it was said that a woman could not go to church to pray or a man to a cigar store for his tobacco without getting some information about rats and plague.

[1] Read at a meeting of the Queensland Branch of the Public Health Association of Australia on November 2, 1921.

What was the result? Operations had begun on September 23, 1907. On March 1, 1909, less than eighteen months afterwards, San Francisco was plague free and, except for a few imported cases, inevitable in a great port, it has remained plague free ever since.

A few years later more extended knowledge of the intimate life details of rats and rat fleas in their capacity of plague carriers enabled the American health authorities in Manila to bring a dangerous plague outbreak under control. Situated only a couple of days' steam from Hong Kong and other Asiatic plague centres, this great native city was constantly open to attack by plague.

The Americans protected their sea-front by absolutely rat-proof concrete wharves, so built that no rat from a vessel could get ashore without swimming. They also fumigated vessels to destroy rats on board. For centres of rat plague occurring ashore they soon realized that the method of sending in men with dogs, traps and poison only drove infected rats all over the vicinity. So, whenever an infected rat was found, they began by setting lines of traps extending out in several directions for a block or two, but avoided any deliberate disturbance of the rats in that particular part, except for a close search for dead rats. All rats found were promptly examined for plague. The furthest points at which infected rats were discovered, were marked on a map and lines drawn connecting them.

The next thing was to build along these connecting lines a close fence of galvanized iron, sunk in the ground so that rats could not burrow under it and too high for them to climb over. Close-fitting gates were provided. Once the fence was completed, they had the rats in that enclosed area at their mercy and a hundred or more men worked over it until no rat survived. Then they made every building rat-proof.

Even then they were not satisfied. They had killed the rats in that particular focus, but they knew that their plague loaded fleas could survive for weeks after the last rat was dead. To detect these fleas they used the well-known method, which was used in Brisbane away back in 1906, of sentinel guinea-pigs. It is a very simple and easy method. Healthy guinea-pigs are made free of fleas and allowed to run in the suspected place for a night or two. Rat fleas like guinea-pigs almost as much at rats and, having no rats to feed on, will soon bite the guinea-pigs. Then the guinea-pig is cleaned of the fleas he has collected, put into a flea-proof cage and watched. If he soon developes plague, it is proof that he was infected in the suspected building. Meanwhile, some of his catch of fleas are crushed up in a little fluid and injected into other healthy guinea-pigs. If they develope plague, it proves that some of the fleas contained plague. Other fleas are examined under the microscope and if they contain plague, the germs are seen. Only when no more infected fleas are found, can the area be regarded as free from infection.

These methods have since been used with success in Japan and in Galveston and other places.

In India, the immense native population, with its extraordinary number of castes and religious sects, has introduced all manner of complications into plague work. One influential sect refuses on religious grounds to permit rat destruction and will fight for the animals, if necessary. Others will not accept protective inoculation, because they suspect that beef is used in making the inoculating material. Plague work there is thus largely influenced by diplomacy and politics. In large centres, such as Bombay, extensive work is done in killing rats and rat fleas in infected premises by the use of hydrocyanic acid gas on a large scale. Inoculation against plague is also carried out extensively, but, generally speaking, although most of the world's modern knowledge of plague control is based on work done by British scientific workers in India, control measures are carried out only on a relatively small scale. It is another illustration of the well-proven fact that when plague control comes up against politics, it is good for plague but bad for the people who suffer from it.

In Central Java the Dutch authorities are fighting plague with success on a vast scale. They base their entire operations on the closely studied habits of two species of rats and one species of rat flea. As with all other successful plague control measures, the ultimate object is to prevent rat fleas from sucking up plague bacilli with the blood of infected rats and pumping them into the bodies of human beings. To attain this result, the Dutch authorities have had to reconstruct some 400,000 native houses and to establish an extensive system. But, when the plague staff moves on, there is no plague left, by reason of the fact that rats cannot exist in the reconstructed houses. No rats means no rat fleas and hence no plague.

Successful plague measures all over the world depend on this one principle. Rats are the originators and conveyors of the disease; therefore, when plague occurs the first object is to confine them as far as practicable to each focus of infection as it is located and to destroy them there as rapidly and thoroughly as possible. Merely to attack them with dogs, traps and poison is risky, for it drives them to escape to other areas. Once destroyed, they must be built out so that they cannot return. Their fleas are the conveyors of plague from rats to human beings, therefore the measures used must be able to destroy fleas as well as rats. Fleas are harder to reach, therefore it must be ascertained by means of guinea-pigs used as flea traps whether and when the premises are safe for occupation; and flea killing measures must be persisted in until they are safe. To neglect the infected fleas means failure, for they will live and remain capable of infecting rats and human beings for at least several weeks after all rats are got rid of.

The principal difficulties experienced during the last decade in other countries in successfully controlling plague have arisen from interference, political or otherwise, with the extensive and careful work which alone can confer protection. Where interference is absent and where a white community buckles to the task as an urgent civic need—as they did in San Francisco and Galveston—the result is

assured, although it needs time and much money. But where interference, slackness, delay and misdirected effort have been allowed to persist and where an apathetic public has permitted itself to be lulled into a false sense of security concerning the terrible possibilities of an ineffectively controlled plague outbreak, the only results to be expected are heavy loss of life and self-protective action by other countries, irrespective of the commercial interests of the country which has failed to protect itself.

PALPABLE RADIAL ARTERY.

By G. C. Willcocks, M.C., O.B.E., M.B. (Syd.), M.R.C.P. (Lond.),
Assistant Physician, Sydney Hospital; Assistant Physician, Coast Hospital.

THE following notes present a *résumé* of the results of an examination of 181 men for the purpose of determining whether the radial artery was or was not palpable. One hundred and four healthy soldiers and 77 ex-soldiers suffering from neurasthenia were examined.

The signs used in the summary of results are:

— indicates that the radial artery was not palpable.

\+ indicates that the radial artery was palpable.

++ indicates a markedly palpable radial artery.

TABLE I.—ONE HUNDRED AND FOUR HEALTHY SOLDIERS.

Number.	Artery.	Infectious Disease	Blood Pressure (mm., Hg.)	Age: Under 26.	Age: Over 26.	Age: Over 36.
74	+	41	134	36	27	11
17	—	7	136	9	—	—
13	++	7	141	4	5	4

TABLE II.—SEVENTY-SEVEN NEURASTHENIC EX-SOLDIERS.

Number.	Artery.	Infectious Disease	Blood Pressure (mm., Hg.)	Age: Under 26.	Age: Over 26.	Age: Over 36.
40	+	15	132	8	19	13
6	—	—	128	—	3	3
31	++	10	134	3	16	12

Of 104 soldiers between the ages of 18 and 50 (average 27) the radial artery was palpable in 85%; in 15% the artery could not be distinguished from the surrounding tissues, *i.e.*, was not palpable (in six of these a thick forearm or proximity of the vessel to the flexor tendons prevented a definite conclusion as to whether the vessel could be palpated after the radial pulse was obliterated or not).

In 13% of the total number the artery felt very thick and hard (markedly palpable, ++).

Of 77 neurasthenic patients, 92% had palpable radial arteries and in 8% the artery could not be felt.

Of the 92% with palpable radial arteries, in 40% the artery felt much thickened (++).

In a number of the men both radial arteries were felt. Some showed slightly greater apparent thickening of the right radial, but in the majority there was no obvious difference between the two sides.

Factors Which Might Influence the State of the Arterial Coats.

I.—Personal Equation in Examination.

It was clearly recognized that clinicians often disagree in determining whether or not a radial artery is palpable. In order to obviate mistakes on this ground the opinion of other practitioners was obtained in as many cases as possible. In none of the patients in whom the author thought that the radial artery was palpable, was this opinion controverted by his colleagues.

II.—Infectious Diseases.

Of 87 soldiers whose arteries were palpable or markedly so, 48 had had some infectious disease since childhood (the majority could not recall their childish complaints).

Of 71 neurasthenic patients with palpable radial artery, 25 had had infectious disease at intervals of from three months to twenty years before examination. Typhoid fever, malaria, scarlet fever, trench fever, influenza, rheumatic fever, pneumonia were the diseases mentioned.

III.—Age.

Of 87 soldiers with palpable radial arteries, 40 were under 26 years.

The artery was markedly palpable in healthy soldiers of 18, 19 and 22 years and was palpable in two soldiers of 18, five of 19, nine of 20 and five of 21 years.

IV.—Alcohol.

In only three men was there a history of excessive indulgence in alcohol. These three men had palpable radials.

V.—Nutrition.

The majority of the neurasthenic patients were thin. The soldiers were healthy young men in the main, with no undue leanness or corpulence.

In some men with thick forearms and in some in whom the artery was close to the flexor tendons, it was difficult to determine whether the radial was palpable or not. These, however, were a very small minority and have been classed as having arteries not palpable (—).

VI.—Occupation.

Two painters and one plumber were examined; all had palpable radials.

Twenty-one of 31 neurasthenics were distinctly palpable arteries had done heavy work as labourers, farmers, shearers, etc.; six had been doing sedentary work as clerks, accountants, etc., before enlistment. Of 40 neurasthenics with palpable arteries, 25 had done heavy work and 14 had followed sedentary occupations. In these statistics the heavy workers (*i.e.*, those who were not employed in sedentary occupations) predominated, there being only 15 sedentary workers among the 77 neurasthenic patients examined.

It does not seem likely that heavy work could have been responsible for the state of the radial artery in 25 healthy soldiers under 22 years whose arteries were palpable.

VII.—Stress and Strain.

Many neurasthenics had undergone much mental strain, both before and since the onset of their symptoms.

The majority of the soldiers had not seen active service; there did not appear to be any difference between the radial arteries of those who had and those who had not seen active service.

Several of the neurasthenic patients, who were much incapacitated by their symptoms and who had been ill for two or more years, looked very old and haggard and presented a very marked thickening of the radial.

It may be of interest to quote one such case as an example of the type alluded to:

An infantry officer, aged 26 in 1920, enlisted in 1914, shortly after leaving school. He was at that time perfectly fit and well; a normal young man.

Captured in 1917, he was taken to Russia, where he was confined with other prisoners for two years in Moscow.

He stated that he passed some months in constant fear of death. He and his companions used to wait up each night between 10 p.m. and 2 a.m. in the expectation of Chinese mercenaries, who were allowed to enter the prison at this time, to murder some of the inmates and appropriate their clothes and possessions.

This officer's hair was grey, his face was lined and haggard and his general appearance that of a man between 40 and 50. His radial artery was very markedly thickened.

VIII.—Hypertonus or Spasm.

It has been stated that contraction of the arterial coats (spasm or hypertonus) sometimes causes the radial artery to be palpable and that after treatment by dieting, rest and iodides the vessel wall becomes soft again.[1]

William Russell attributes many symptoms to hypertonus. He states that the sphygmomanometer does not record blood pressure alone, but records blood pressure *plus* resistance of the arterial wall, which may vary from 5 mm. of mercury to 8 mm. or more. He argues that if the sphygmomanometer reading falls 20 mm. of mercury or more in an individual under treatment, this fall is not due to a fall of blood pressure, but to relaxation of the arterial walls which were formerly hypertonic.

It is difficult to believe that hypertonus could be the cause of the palpable radial artery in so great a number of men as are here reviewed; if this were the cause, it would be interesting to know why so many men have hypertonic arteries.

In three cases an endeavour was made to eliminate possible spasm by immersion of the forearm in water as hot as could be borne for five to ten minutes. The radial arteries were just as easily palpable immediately after this immersion as they had been before immersion.

It will be necessary for this question of hypertonus to be more fully investigated before a definite conclusion can be arrived at as to its effect in causing palpable radial arteries in normal individuals.

Conclusions.

The foregoing observations indicate that the radial artery is palpable in the majority of men after 18 to 20 years of age.

It is more frequently and more markedly palpable in soldiers suffering with neurasthenia, especially in those with marked neurasthenic symptoms of long standing.

It is more easily palpable in thin people.

It is more often palpable in those who have suffered from infectious disease.

In this series it was more often palpable in sedentary workers than in heavy workers.

It does not vary with age up to the age of 50 years; that is to say, it is not necessarily more easily palpable at 45 than at 20.

There is no proof that hypertonus or spasm is the cause of the radial artery being palpable in the majority of men.

Methods.

The method of examination of the radial artery was that suggested by Osler and McCrae.[2]

To estimate the thickness of the vessel wall the pulse wave should be obliterated in the radial and the vessel wall felt beyond.

In a perfectly normal vessel the arterial coats under these circumstances cannot be differentiated from the surrounding tissues, whereas, if thickened, the vessel can be rolled beneath the finger.

Hutchinson and Rainy state that: "In health the vessel wall can rarely be felt unless the arm is thin."[3]

This statement gives no indication as to the proportion of men in whom the artery can be felt.

Sir James MacKenzie says:

We recognize the yielding nature of the arterial coats in healthy arteries; in degeneration of the coats the walls may be universally thickened or contain bead-like patches of induration. . . . .[4]

In these notes a palpable radial artery is one which can be differentiated from the surrounding tissues when the pulse is obliterated, which can be rolled beneath the finger and which does not completely yield to pressure.

Discussion.

In his recent comprehensive treatise on arterio-sclerosis Sir Clifford Allbutt makes no positive statement as to the frequency of palpable radial arteries; he considers that palpable radials are frequent in those who are occupied with heavy physical labour.[5] He quotes (*i.*) Thayer, who found 25% palpable radials between 10 and 20 years, 50% palpable radials between 20 and 50 years, 100% palpable radials between 50 and 60 years, 48.3% palpable radials in 183 persons following typhoid fever, at all ages, from one month to thirteen years after the attack. (*ii.*) Frantzel, who stated that the radial artery was palpable in a considerable proportion of 500 German soldiers after war and exposure in the Franco-German war. (*iii.*) The Dickson brothers, who examined 500 English miners and found that the radial artery was palpable in all except twelve over 20 years of age. The Dicksons attributed this condition to gaseous poisons.

What is the significance of a palpable radial artery? Generally it will be found that text-books of pathology give no idea of the state of the arteries at different ages, apart from certain diseases such as atheroma and arterio-sclerosis.

Although a markedly palpable radial artery is frequently regarded by clinicians as evidence of arterio-sclerosis, it is extraordinary how rarely the radial artery is alluded to in text-books of pathology. Most commonly it is the larger arteries and especially the aorta, that are dealt with.

In a paper dealing with the histological appearances of the radial, aortic and mesenteric arteries Thayer[6] stated that the radial artery became thicker by an increase of connective and muscular tissue in each decade, that it was thicker in the second, third and fourth decades, especially in heavy workers and alcoholics, and in the fifth decade especially with chronic nephritis, aortic regurgitation, heavy work and alcohol. Further, he stated that the radial artery was palpable in the majority (70 odd) of cases studied, but generally not thickened; that changes in the mesenteric artery are similar to those in the radial from year to year and that changes in the radial which are marked, generally indicate similar changes in mesenteric and aortic arteries.

Thayer's researches suggest that when the radial artery becomes palpable or markedly so, it is because of an increase in connective and muscular tissues. If, as Thayer states, changes in the aorta and mesenteric arteries are similar, some degree of general arterio-sclerosis must exist in such an indigeneral arterio-sclerosis must exist in such a case. Very few of the men dealt with in these sclerosis and those who did present tortuous temporal or radial arteries, tortuous and pulsating brachial arteries and high-pitched aortic second sound, were with one exception men between 45 and 50 years of age.

The exception was a young man of 25, who complained of dyspnœa on exertion and whose condition had been diagnosed as "D.A.H." (disordered action of the heart). This patient's pulse gave the impression of high blood pressure at once. His systolic blood pressure was 180 mm. of mercury. This appeared to be a case of "hyperpiesis."[5]

Blood Pressure.

In connexion with the question of arterio-sclerosis in these subjects, it was thought that the blood pressure might be of interest. It is commonly said that blood pressure estimations are reliable, providing they are taken at rest in the recumbent posture, the reason for this being that posture affects blood pressure differently in each individual.

This statement suggests that blood pressure readings are unreliable taken in other positions. However, low or high blood pressure is usually suspected when the pulse is taken in the consulting-room or in the out-patient department with the patient in a sitting or standing position, so that the estimated tension of the pulse is undoubtedly of value in these positions.

The blood pressure of these 181 patients was taken with Lauder Brunton's sphygmomanometer on the right upper arm with the patient in the standing position. The systolic pressure only was taken, as the significance of diastolic and pulse pressure is not so well understood at present.

The summary of results in 104 soldiers is given in groups, the subjects in each group being examined at the same time and under the same conditions.

Group I.—Forty-one healthy men, aged 18 to 30 years (average age 21 years). Blood pressure was taken at 2.30 p.m., following lunch at 12.30 p.m. and the smoking of one or more cigarettes in nearly every case. The men had been lying down between lunch and the examination time. Limits of systolic blood pressure: Lowest, 120 mm. Hg.; highest, 160 mm. Hg.; average, 144 mm. Hg..

Group II.—Seventeen men, average age 31 years, at 2.30 p.m. after moderate work at "stables." Lowest blood pressure, 115 mm. Hg.; highest blood pressure, 180 mm. Hg. (a case of hyperpiesis); average blood pressure, 142 mm. Hg..

Group III.—Thirty-three men, average age 33. Blood pressure was taken at 1.30 p.m.. All had vigorous exercise in the morning (physical training), lunch at 12.30 p.m., followed by a rest. A hot, muggy day; much sweating. Lowest blood pressure, 110 mm. Hg.; highest blood pressure, 145 mm. Hg.; average blood pressure, 123 mm. Hg..

Group IV.—Thirteen men, average age 31. Blood pressure taken at 11 a.m., ten minutes after firing practice with a 23.4 cm. (9.2 inch) naval gun. Lowest blood pressure, 110 mm. Hg.; highest blood pressure, 160 mm. Hg.; average blood pressure, 132 mm. Hg..

Group V.—Seventy-seven neurasthenic soldiers. Blood pressure taken between 3 p.m. and 4 p.m. after resting. Average age, 34. Lowest blood pressure, 100 mm. Hg.; highest blood pressure, 160 mm. Hg.; average blood pressure, 133 mm. Hg..

It will be seen by reference to Tables I. and II. that the average blood pressure differed very little in those whose arteries were markedly palpable, just palpable or not palpable at all and that there was a slightly higher reading on the average among the healthy than among the neurasthenic soldiers, though the age of the latter on the average was 34, as compared with healthy soldiers, whose average age was 27.

If, disregarding averages, the individual blood pressure readings are examined, it is found that they do not vary according to the degree of palpability of the radial artery, nor do they vary with the age of the subject at all constantly. There are many readings of 115 to 120 mm. of mercury in men between the ages of 40 and 50 and a number over 140 mm. of mercury in men of 20 and 21 years.

Curiously enough, Group I. (the youngest men) presented the highest average blood pressure, 144 mm., at 21 years of age. Nearly all these men smoked one or more cigarettes shortly before examination.

The men of Group II., whose average age was 31 and average blood pressure 142 mm. of mercury, had undergone hard training in camp the previous week and had been doing moderate work just before examination.

The men of Group III. had done hard physical training in the morning, running, physical exercises, etc.; it was a hot day and the men were sweating even at rest. The sweating may have accounted for the low average blood pressure in these cases—123 mm. of mercury at the age of 33.

Group IV. was expected to show some evidence of increased blood pressure, as the men in this class had been engaged in fairly vigorous and stimulating work. Usually the firing of a heavy gun produces

some degree of tension or excitement in those who are within a few metres of it and it was thought that this excitement might produce some stimulation of the adrenals through the sympathetic, with consequent increase of adrenalin in the blood and rise of blood pressure.[7]

Allbutt states that the blood pressure is raised by excitement for ten to sixty minutes. In this group the average blood pressure was 132 mm. (average age 31). There was therefore no evidence that the blood pressure of these men was increased to any extent. The average blood pressure in males before middle age is estimated at 115 to 150 mm. of mercury.[5] The average blood pressure in all these healthy soldiers was 110 to 160 mm. of mercury.

Conclusions.

1. The sphygmomanometer reading in the brachial artery in healthy men (standing) varies between 110 and 160 mm. of mercury.
2. It does not vary with age definitely.
3. It is not necessarily greater in men whose radial arteries are palpable than in those whose radial arteries cannot be felt.
4. It is lower two to three hours after exertion and sweating.
5. It is raised by smoking.
6. It is not found to be raised ten to twenty minutes after excitement.

In concluding these notes, I should like to thank Sir Humphry Rolleston for much sound advice and kind encouragement in connexion with the work and to thank Colonel Eames, Dr. Noble and Dr. Carruthers for their great assistance in enabling me to carry out these observations.

References.

(1) "The Sphygmomanometer," William Russell, 1920.
(2) "The Principles and Practice of Medicine," Osler and McCrae, 1920.
(3) "Clinical Methods," Hutchinson and Rainy.
(4) "Diseases of the Heart," James Mackenzie.
(5) "Diseases of the Arteries," Clifford Allbutt.
(6) "Studies on Arterio-Sclerosis," W. S. Thayer and Marshall Fabyn, *American Journal of Medical Sciences*, 1907.
(7) "Principles of Human Physiology," Starling.

A NOTE ON LUMBAR PUNCTURE AS A DIAGNOSTIC AND THERAPEUTIC MEASURE.

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Introduction.

THE object of this note is to demonstrate briefly the use of lumbar puncture in diagnosis and its undoubted value as a therapeutic measure.

The recent experimental work of Webster[1] on monkeys led him to the conclusion that lumbar puncture should be reserved for use in those cases in which "definite clinical signs of meningitis are present" and he "deprecates its use as a royal road to the diagnosis of an obscure condition."

Lumbar puncture has been employed of recent years much more frequently, both in diagnosis and treatment, than heretofore. Reports of ill-effects following the procedure are not frequent. No less than 4,000 cases were reported from an American depôt without any undesirable after-effects. French's "Index of Differential Diagnosis of Main Symptoms" (1920) gives much prominence to lumbar puncture as a diagnostic procedure, even in the section on "Prolonged Pyrexia" (by Sir Frederick Taylor), which includes the differential diagnosis of typhus, typhoid and paratyphoid fevers and the like. No mention is made of consequent meningeal symptoms.

Webster's experiments are very suggestive. My clinical experience does not confirm them completely. That lumbar puncture may sometimes precipitate meningitis in severe types of septicæmia is true and the procedure is therefore in those cases attended with risk. In cases of meningism, as shown below, such has not been the case. In obscure cases, with or without suspicion of infections such as typhoid fever, pneumonia, etc., I have used lumbar puncture, repeated, if thought advisable, as a routine procedure in the diagnosis of dozens of cases during the last three years without observing any undesirable after-effects attributable to its use. In every instance the patient was in hospital and so allowed the course of the illness to be followed to its conclusion. Such being the case, I cannot support, on clinical evidence, Webster's conclusion "that the lumbar puncture needle should not be employed except in the presence of definite clinical signs of meningitis."

It must be remembered, however, that the normal pressure of the cerebro-spinal fluid is such as to cause it to flow from the needle at the rate of one drop per second. When this is found to be the case, it is hardly necessary to say the needle should be withdrawn at once. I believe that good results are often obtained by withdrawal of an excess of cerebro-spinal fluid due to the pathological conditions, but I have seen two cases of fatal syncope caused very probably by drainage of cerebro-spinal fluid from an intra-thecal space in which the pressure was normal.

The chemical and microscopical examination of the cerebro-spinal fluid may indeed throw a flood of light upon obscure conditions and so make a diagnosis possible at an early stage of the disease, when immediate treatment will have its greatest effect.

As a therapeutic measure, lumbar puncture, repeated at intervals or performed once, as the symptoms indicate, is of undoubted value in many cases.

Syphilis of the Central Nervous System.

Cerebro-spinal syphilis, which manifests itself in such a variety of clinical guises, is notoriously difficult to diagnose in its earliest stage. Vague pains, a change in temperament or personality, slight paræsthesia and numerous other apparently trivial complaints may be the first intimation, if recognized as such, of a luetic lesion of the central nervous system. In many cases the blood fails to give a Wassermann reaction, even after a provocative injection of an arsenical preparation. In a case of this kind, which is by no means rare, a lumbar puncture revealing fluid under increased pressure is the only method of making an immediate diag-

[1] THE MEDICAL JOURNAL OF AUSTRALIA, November 27, 1920.

nosis. When the cerebro-spinal fluid is found to contain an increased number of lymphocytes per cubic millimetre and to react to Wassermann's or to the colloidal gold test, a sure diagnosis can be made which otherwise would have been impossible. The frequent absence of the earlier lesions (skin, etc.) of syphilis in these cases (probable infection by a neurotropic strain of spirochæte) and the paramount importance of an early diagnosis for success in treatment still further enhance the value of the information obtained by lumbar puncture. Even should the procedure show a normal cerebro-spinal fluid, negative evidence in diagnosing obscure cases is not unworthy of attention.

It is my practice in treating syphilis of the central nervous system to perform lumbar puncture at intervals of a few weeks to estimate the number of cells (lymphocytes) per cubic millimetre in the cerebro-spinal fluid as an indication of the effect of treatment, as is done in the Mayo Clinic and other hospitals.[1]

Encephalitic Lethargica.

Encephalitis lethargica is another disease which may vary considerably in its onset and clinical course and so cause real difficulty in diagnosis. The majority of the dozen cases which have been under my own care, conformed to the lethargic type. Clear cerebro-spinal fluid under pressure, with few or no lymphocytes, was present in every case and a link in the chain of evidence necessary to arrive at a correct diagnosis.

These cases were treated with rest in bed, in a quiet room, when possible, attention to bowels, etc., drainage of the excess of cerebro-spinal fluid by means of repeated lumbar punctures and the exhibition of urotropine by mouth in some cases.

There was one fatal case. The patient had been ill for two weeks before admission and had had no treatment. Death occurred two days after admission.

In every case lumbar puncture was performed immediately the diagnosis was made. The fluid was allowed to escape until there was a marked reduction in pressure. The average amount withdrawn at each puncture was 30 c.cm., the minimum 6 c.cm. and the maximum 50 c.cm.. Lumbar puncture was repeated every twenty-four hours till the symptoms abated. The minimum number of times lumbar puncture was performed in any individual case was three, the maximum ten and the average eight. In all but one instance the relief of headache and lethargy was marked after withdrawal of the fluid. During the height of the disease the symptoms (headache and lethargy) tended to become worse towards the end of the twenty-four hours and were relieved by repetition of the lumbar puncture. The fluid became less at each puncture, the pressure less and the symptoms abated as the amount of fluid decreased. The relief of the main symptoms (headache and lethargy, the ocular symptoms being always early and transient) indicated the cessation of the treatment, although frequently the cerebro-spinal fluid was still under considerable pressure when this happened, but never as great as in the more acute stage of the attack.

In one case, the first of the series, which was particularly severe, with delirium and high fever, after an interval of eleven days without treatment and free from symptoms, lethargy and headache returned. Withdrawal of 48 c.cm. of fluid by lumbar puncture restored the patient to normal within twenty-four hours. Six days later these symptoms re-appeared. Again the withdrawal of 40 c.cm. of cerebro-spinal fluid promptly relieved the condition. The patient had had no recurrence of symptoms when last seen six months after his illness.

I have had no opportunity of comparing this series with other cases treated without drainage of the cerebro-spinal fluid; but in my hands the procedure has never failed to give relief and in one case only failed to give marked relief. The mortality rate also in this series was comparatively low—8.3%. The series was too limited in number to allow of far-reaching conclusions, but I think I am justified in claiming that repeated lumbar puncture was an important factor in recovery.

Meningism.

The classical signs, head retraction, more or less marked Kernig's sign, etc., are usually the first intimation of meningeal involvement.

A boy, aged 6 years, suffering from an acute attack of measles, suddenly developed head retraction, slight confusion of mentality and a marked Kernig's sign. Lumbar puncture was performed immediately and 20 c.cm. of fluid withdrawn. The fluid was under considerable pressure.

The first specimen contained some red blood cells and a few polymorpho-nuclear pus cells. No organisms were seen.

In the third specimen there were also red blood cells and a few polymoropho-nuclear pus cells, but no organisms.

Lumbar puncture was repeated daily for seven days and 20 c.cm. to 35 c.cm. of fluid withdrawn on each occasion.

All signs of meningeal irritation had then disappeared. Whether this case should be classed as meningism or as meningitis, the only treatment was repeated lumbar puncture.

There is, of course, no evidence that lumbar puncture shortened the duration of the meningeal symptoms.

The obvious improvement in the child's mental condition and comfort immediately after the first lumbar puncture convinced me that the treatment was beneficial.

I have had a similar experience in two other cases of meningism in children, one during the course of a lobar pneumonia, in which clear fluid under increased pressure containing no cell elements was withdrawn, and the other in a child convalescent from typhoid fever. The meningism commenced after the temperature had been normal for one day and did not cause a further rise.

Lead Encephalopathy.

A colleague kindly allowed me to read some notes which he made while resident medical officer in the

[1] *Medical Clinics of North America*, November, 1919, and May, 1920.

Children's Hospital, Brisbane. The notes emphasized the prevalence of lead poisoning at that time and the fact that convulsions were a frequent complication. Lumbar puncture and withdrawal of cerebro-spinal fluid which was always under increased pressure, was found to give excellent results, relieving the main symptoms of the encephalitis almost at once.

Hemiplegia and Epilepsy.

General paralysis of the insane may attract no attention in a patient till there is a sudden seizure of some kind, frequently epileptiform and sometimes producing a hemiplegia like that of a cerebral hæmorrhage. The result of a Wassermann test applied to the cerebro-spinal fluid and the presence or absence of lymphocytes in the cerebro-spinal fluid may point to the true nature of the case.[1]

Coma.

In all cases of coma in which diagnosis is in doubt, lumbar puncture is indicated (Thomson and Miles).

I have seen uræmic coma and convulsions relieved on three occasions when other measures had failed. Excess of urea, phosphates and sulphates, with a decrease of chlorides in the cerebro-spinal fluid, is stated to be of diagnostic importance in uræmia.

In patients admitted to my wards in an unconscious condition, it is my custom, when the diagnosis is in doubt, to perform a lumbar puncture as a routine procedure. I have never regretted it. The information gained by an examination of the fluid has on several occasions enabled me to commence treatment at once, backed by an almost certain diagnosis.

Other Conditions.

There is no need to do more than mention the value of lumbar puncture in the diagnosis of the following diseases: Tuberculous meningitis, cerebro-spinal fever (due to *Diplococcus meningitidis*, staphylococcus or other organisms), fracture at the base of the skull (blood in cerebro-spinal fluid), cerebral abscess, especially consequent to *otitis media*, intra-cranial and intra-spinal hæmorrhage, paraplegia and hemiplegia.[1]

In some cases of cerebral and spinal tumours lumbar puncture is of value. Aberrant (cancer)

[1] French: "Index of Differential Diagnosis of Main Symptoms."

[1] French: *Loc. cit.*.

Case of Cerebro-Spinal Fever with Recovery.

J.P., aged 30 years, a labourer, was admitted to the infectious block of the Newcastle Hospital on November 11, 1920. He exhibited the classical signs and symptoms of cerebro-spinal fever.

Date.	Temperature.	Pulse.	Amount of Cerebro-Spinal Fluid Withdrawn.	Amount of Serum Injected.	Remarks.
November 11, 1920	37.2° C.	80	70 c.cm.	30 c.cm.	Fluid turbid and under pressure; numerous pus cells and intracellular diplococci
November 12, 1920	37.2° C.	75	50 c.cm.	30 c.cm.	———
November 13, 1920	37.2° C.	70–78	60 c.cm.	30 c.cm.	Fluid as above; pus cells increased
November 14, 1920	37.2° C.	88	70 c.cm.	30 c.cm.	———
November 15, 1920	37.6° C.	80	75 c.cm.	45 c.cm.	———
November 16, 1920	36.8° C.	75	75 c.cm.	45 c.cm.	Fluid as above; pus cells increased
November 17, 1920	37.8° C.	75	60 c.cm.	45 c.cm.	———
November 18, 1920	37.8° C.	100	100 c.cm.	30 c.cm.	Patient delirious; strabismus; inclined to be violent; passing fæces and urine involuntarily; critical condition
November 19, 1920	38.4° C.	95	90 c.cm.	30 c.cm.	———
November 20, 1920	37.8° C.	110	75 c.cm.	30 c.cm.	Pathological report on fluid: Red blood cells 5,000 per c.mm.; white blood cells, 1,800 per c.mm.; pathological increase of cells, 1,772 per c.mm.
November 21, 1920	38.4° C.	100	90 c.cm.	30 c.cm.	No more serum given. The pathological report showed that the serum was having no effect in decreasing the number of cells and organisms in the cerebro-spinal fluid. It was not a polyvalent serum, *i.e.*, was not made from various strains of the *Diplococcus meningitidis*.
November 22, 1920	37.9° C.	120	75 c.cm.	——	———
November 23, 1920	36.8° C.	120	95 c.cm.	——	———
November 26, 1920	——	—	65 c.cm.	——	———
November 27, 1920	——	—	30 c.cm.	——	———
November 28, 1920	——	—	50 c.cm.	——	———
					Recovery was complete. The strabismus entirely disappeared.

As the serum had been administered intrathecally for ten days and the patient had become progressively worse, we decided that its further use was contra-indicated. The pathologist's examination of the cerebro-spinal fluid on November 20, 1920, did not indicate any effective combating of the organisms by the serum. We anticipated a fatal termination, but persisted with spinal drainage, with the result as shown in the table.

cells have been described in this connexion. In a case of convulsions of sudden onset and uncertain cause and in cases of cerebral vomiting lumbar puncture as a routine step will often lead to a correct diagnosis and save valuable time. Jex-Blake,[1] in discussing convulsions as the initial symptom of cerebral abscess and cerebral tumour, says: "It may happen that an epileptiform fit, with unilateral or bilateral convulsions, is the first sign that anything is wrong, or at any rate the first thing that makes the patient consult a medical man."

Here, again, lumbar puncture may be necessary for a speedy, correct diagnosis.

The record of a case of cerebro-spinal fever with recovery is of interest, on account of the part played by spinal drainage in the treatment. The case is not reported in detail. A few notes and a table recording the results of repeated lumbar puncture have been deemed sufficient for the purpose of this note (see previous page).

Lésne[2] reports a case of paratyphoid meningitis treated in a similar manner with a favourable result.

In the cases quoted above the fluid was allowed to run till the pressure began to diminish and the needle was removed while the fluid was still running freely.

The amount to be removed was judged by the initial pressure and the diminution of the rate of flow.

Reports of Cases.

AMŒBIC ABSCESS OF THE LIVER.

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Introduction.

AMŒBIC abscess of the liver is not infrequently the source of diagnostic errors, especially in countries where amœbic dysentery is not endemic. The following two cases illustrate some of the difficulties and emphasize some interesting points in the surgical pathology of this disease. The condition is not common in Australia and most of the cases occur in patients who have at one time resided in the tropics, where amœbiasis is endemic. There have, however, been some cases reported in which the disease appeared to be indigenous to Australia.

The following are all the cases which I have been able to trace: Fiaschi and Isbister recorded one case in Sydney in 1898,[1] Isbister reported four cases in New South Wales in 1899,[2] Verco added two from Adelaide in 1899[3] and Corlette reported one in 1915 from Maitland, New South Wales.[4]

The four patients of Dr. Isbister's series appear to have been infected by means of their water supply from another miner who was operated on in Sydney for a liver abscess of unknown ætiology.

The Australian troops in the Near East war zone suffered from the ravages of amœbic dysentery and, when we consider the insidious nature of amœbiasis and the special proneness of the white races to this particular manifestation of the disease, we must expect the development of amœbic abscess in some of these returned soldiers.

[1] French: *Loc. cit.*.

[2] Lésne: Abstract in "Epitome of Current Medical Literature," *British Medical Journal*, November 27, 1920.

Case No. 1.

M.S., male, aged 40 years, was admitted with a diagnosis of pneumonia on January 1, 1921.

Present History.—Three months ago he had a chill and a rigor and a severe cough developed. He has had pain in the chest, expectoration, but no hæmoptysis. He has been "off colour" all this time and has lost 19 kilograms in weight. On admission, his temperature was 39.4° C., his pulse-rate 100 and his respirations 32.

Past History.—Twenty years ago, while living in Victoria, he had an attack of so-called dysentery, but it was not severe enough to keep him in bed. Fifteen years ago he was in South Africa for a few months. He had no dysentery there, but there was severe dysentery on board the boat he travelled on.

Examination revealed dulness at the base of the right lung, with diminished breath sounds. His illness was regarded as pneumonia and was treated as such.

On January 4, 1921, there was much blood-streaked sputum with muco-pus. No tubercle bacilli were found.

On January 5, 1921, X-ray examination showed diminution of translucency of the base of the right lung and some limitation of diaphragmatic movement.

For the three weeks up to January 24, 1921, the patient had a high, swinging temperature, coughed up a great quantity of muco-pus tinged with blood and on that date still showed the same signs in his chest as before. Pfeiffer's bacillus was isolated and he was treated with a vaccine which caused some reaction.

On January 18, 1921, some tenderness was noticed in the right hypochondrium. No complement deviation test for hydatid was elicited.

An X-ray examination was carried out on January 28, 1921, but no abscess was detected and partial consolidation was no longer seen.

He was treated with intravenous injections of Pfeiffer's bacillus vaccine with temporary benefit. The sputum was still blood-streaked and pinkish and contained muco-pus on February 1, 1921. Since January 26, 1921, pleural crepitus had been noticed on the right side and the chest condition was becoming worse.

On February 9, 1921, there was extension of crepitus and dulness in the right side of the chest into the axilla. The leucocyte count was 30,000.

On February 12, 1921, the right pleural cavity was aspirated; 60 c.cm. of blood-stained fluid was removed.

On February 16, 1921, diarrhœa commenced and became very troublesome; amyloid disease was suggested. Bronchoscopy revealed contracted bronchi; there was no bronchiectasis.

On February 19, 1921, the diarrhœa was still troublesome. *Entamœba histolytica* was present in large numbers in the stools. The sputum was unchanged, but the lung involvement was more extensive. Some tenderness in the liver area was detected.

An operation was performed on February 21, 1921. When the pleura was opened, a quantity of turbid fluid escaped. A tube was inserted into a subphrenic collection of pus. A course of emetine (0.06 grm.) was ordered for twelve days.

On March 5, 1921, the patient had drained a great amount of pus, but was steadily becoming worse.

On March 8 his general condition was much worse and the presence of a further collection of pus was suspected.

He died on March 10, 1921.

Autopsy revealed general peritonitis.

The lung of the right side was adherent to the diaphragm and on section an abscess cavity was found in the base of the lung continuous with an abscess which had apparently been primary in the upper part of the liver. A communication existed into a large bronchus and in the wall of the cavity were many large bronchi compressed together. Evidently the abscess had been a very large one, but had been evacuated as sputum and had partially collapsed. There were also two small, localized interlobar empyemata, each containing about 30 c.cm. of pus. The colon showed a healthy, granulating surface, demonstrating well the therapeutic action of emetine. (See Figure I..)

Case No. 2.

O.F., male, aged 38 years, was admitted with the diagnosis of cholelithiasis.

Present History.—The patient's illness commenced with

abdominal pain eight days previously. These pains gradually localized themselves to the right hypochondrium. Later the pain became associated with pleuritic pain and when he was admitted, pleural crepitus was present. On admission his temperature was 38.9° C., his pulse-rate was 104 and his respirations 32.

Past History.—The patient had been all over the world, had lived in the tropics and had had dysentery, though never very severely.

An operation was carried out on March 17, 1921. A Mayo-Robson incision was made. Free fluid was found in the peritoneal cavity. The liver was enlarged and when some infraphrenic adhesions were separated, pus welled out. The ninth rib was resected in the posterior axillary line and transpleural drainage of the abscess instituted.

No amœbæ were found in the pus on March 18, 1921, but the patient was put on a course of emetine.

On March 25, 1921, the base of the right lung was explored and pus was obtained.

On March 29, 1921, he had been coughing up large quantities of brownish muco-pus for some days. On this date a further operation was carried out and a portion of the eighth rib was resected and about a litre of pus removed.

He died on April 1, 1921.

Autopsy revealed a subdiaphragmatic abscess, a large abscess of the right lobe of the liver, multiple abscesses of the lung and empyema. The caecum contained a large, deep ulcer, 3.8 cm. by 1.25 cm., with a dirty, sloughing base and from it active *Entamœbæ histolyticæ* were obtained. The rest of the colon showed a little thickening, but was free of ulceration. Microscopical section of the ulcer showed many amœbæ in the submucosa. (See Figure II..)

Conclusions.

Both these cases illustrate the following points in the surgical pathology of amœbiasis:

(*i.*) There may be a very long latent period between the probable primary infection and the development of the abscess, *e.g.*, in Case No. 1, a period of fifteen years.

FIGURE I..
A: Abscess cavity opening into a bronchus.
B: Compressed bronchi in wall of abscess.
C: Interlobar empyema.
D: Thickened and adherent diaphragm.

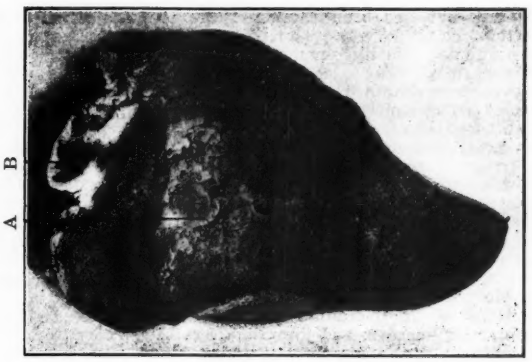

FIGURE II..
B: Spread to subdiaphragmatic area.
A: Amœbic abscess of liver.

(*ii.*) There may be no history at all of an attack of dysentery, as in Case No. 1, and this is said to be true of 10% to 15% of cases. Thus liver abscess may be the first and perhaps the only manifestation of amœbiasis.

(*iii.*) The dramatic appearance of diarrhœa in Case No. 1 and the total absence of diarrhœa in Case No. 2, even in the presence of a definite ulcer of the colon, demonstrates how latent the colonic infection may be.

(*iv.*) Both these cases illustrate the fatal results of secondary infection and show the typical manner of spread to the lung and peritoneum.

(*v.*) In Case No. 1 the absence of hepatic enlargement and the negative X-ray findings were due to the fact that the greater part of the abscess had been evacuated through the bronchi.

(*vi.*) Further, they should teach us to suspect this disease in any case of obscure suppuration in the upper part of the abdomen and to carry out examinations of the fæces as a routine measure. In both these cases either cysts or vegetative forms of the *Entamœba histolytica* would undoubtedly have been found soon after admission, had the fæces been carefully examined.

These cases were under the care of Mr. Langlands and Mr. Upjohn respectively and I am indebted to them for permission to report them.

References.

(1) Fiaschi, T. H., and Isbister, J. L. T.: *Australasian Medical Gazette*, December, 1899, page 511.
(2) Isbister, J. L. T.: *Transactions of the Intercolonial Medical Congress*, Brisbane, 1899, Volume I., page 82.
(3) Verco, J. C.: *Australasian Medical Gazette*, February, 1899, page 66.
(4) Corlette, C. E.: THE MEDICAL JOURNAL OF AUSTRALIA, May 8, 1915, page 427.

Reviews.

GENERAL PATHOLOGY.

THE text-book of general pathology by Drs. J. Martin Beattie and W. E. Carnegie Dickson, first published in 1908 and followed in 1909 by the companion text-book of special pathology, was welcomed as a fitting expression of the teaching of the Edinburgh School, particularly under the influence of the late Professor W. S. Greenfield, who placed the notes of his lectures at the disposal of the authors. A second edition of the volume on general pathology, now issued,[1] preserves the special characters of the

[1] "A Text-Book of General Pathology for the Use of Students and Practitioners," by J. Martin Beattie, M.A., M.D., M.R.C.S., L.R.C.P.; and W. E. Carnegie Dickson, M.D., B.Sc., F.R.C.P.; Second Edition, 1921. London: William Heinemann (Medical Books), Limited; Royal 8vo., pp. 496, illustrated by 232 figures. Price: 31s. 6d. net.

original, but has been thoroughly revised, many of the sections being largely re-written. A larger format has been adopted and the number of pages has been increased by over fifty. The excellent quality of paper and print has been maintained and the fine illustrations, which formed so conspicuous a feature of the original edition, have been largely reinforced.

A clear and up-to-date statement is given of the main doctrines of general pathology. Bacteriology is excluded, as more properly treated in separate manuals. A brief chapter on immunity is included. There is much room for doubt whether the resulting detachment from bacteriological considerations is wise, but doubtless questions of bulk and cost weighed with the authors. It may be suggested that the avoidance of systematic bacteriology is compatible with a brief but adequate emphasis on the main relations of micro-organisms to disease processes. The chapter on necrosis and gangrene has been re-written, so as to include new information gained from experience in the war. That on embolism is of special excellence and is a record of much valuable original work. Under "Inflammation" a good account is given of the cells which appear in the exudates and in the tissues, including the work of Maximow, Marchand and Beattie. Phagocytosis is adequately treated and the section on "Repair" has been enriched by excellent drawings taken from the gold medal thesis on "Inflammation" by Dr. James Dawson, of the Laboratory of the Royal College of Physicians of Edinburgh. In the study of neoplasms, Adami's classification is followed, with special treatment of endothelioma, mesothelioma and perithelioma. The section on animal parasites continues to be a striking feature of the book, with copious illustration, and has been carefully revised, so as to include the most recent knowledge. A useful new chapter on fever has been added. The sections on chemistry have been revised by Dr. John Milroy, of Queen's College, Belfast, and the notes concerning the suprarenal glands have been supplemented by Professor Ernest Glynn, of Liverpool University.

The new colour plates include the cells of inflammatory exudates, inflammation and repair and the life cycle of malarial parasites. New illustrations in the text enrich the sections dealing with degenerations, necrosis, venous congestion, inflammation and abscess, syphilis of spleen and lung, actinomyces and malignant pustule, tumours, pathogenic protozoa, the life history of *Schistosoma hæmatobium* and *Schistosoma mansoni* and also of anchylostoma; temperature charts are shown of several typical forms of fever.

Altogether, this new edition may be highly commended as a sound, reliable and attractive statement of the main doctrines of general pathology.

POST-GRADUATE COURSE IN SYDNEY.

THE following programme has been arranged by the Sydney University Extension Board for the post-graduate course in medicine to be held in January, 1922:

January 9, 1922.

9.30 a.m. to 11 a.m.: Inaugural Lecture, by PROFESSOR A. E. MILLS, at the Royal Prince Alfred Hospital.
11 a.m. to 12.30 p.m.: Cardio-Vascular Diseases, by DR. G. E. RENNIE, at the Royal Prince Alfred Hospital.
Afternoon: Demonstrations in the wards and theatres of the Royal Prince Alfred Hospital.

January 10, 1922.

9.30 a.m. to 11 a.m.: Respiratory Diseases, by DR. S. A. SMITH, at the Royal Prince Alfred Hospital.
11 a.m. to 12.30 p.m.: Acute Abdominal Diseases, by PROFESSOR F. P. SANDES, at the Royal Prince Alfred Hospital.
Afternoon: Demonstrations in the wards and theatres of the Royal Prince Alfred Hospital. Demonstrations in the Pathological Department of the Royal Prince Alfred Hospital, by DR. A. H. TEBBUTT.
Evening: Demonstration in the Tuberculosis Dispensary at the Royal Prince Alfred Hospital.

January 11, 1922.

9.30 a.m. to 11 a.m.: Medical Diseases of Children, by DR. J. MACDONALD GILL, at the Royal Alexandra Hospital for Children.
11 a.m. to 12.30 p.m.: Surgical Diseases of Children, by DR. R. B. WADE, at the Royal Alexandra Hospital for Children.
Evening: Demonstrations of the Modern Treatment of Venereal Diseases at the Royal Prince Alfred Hospital.

January 12, 1922.

9.30 a.m. to 11 a.m.; Lecture on the Diseases of the Digestive System, by DR. H. HAMILTON MARSHALL, at the Sydney Hospital.
11 a.m. to 12.30 p.m.: Operative Surgery of the Large Bowel, by SIR HERBERT L. MAITLAND, at the Sydney Hospital.
Afternoon: Demonstrations in the wards and theatres of the Sydney Hospital.
Evening: X-Ray Demonstrations of the Digestive Tract, by DR. J. G. EDWARDS, at the Sydney Hospital.

January 13, 1922.

9.30 a.m. to 11 a.m.: Acute Pelvic Affections, by DR. GEORGE ARMSTRONG, at the Sydney Hospital.
11 a.m. to 12.30 p.m.: Cardio-Vascular Diseases, by DR. H. J. RITCHIE, at the Sydney Hospital.
Afternoon: Demonstrations in the wards and theatres of the Sydney Hospital.

January 16, 1922.

9.30 a.m. to 12.30 p.m.: Principles of Treatment and Demonstrations of Fractures of the Lower Limb, by DR. C. E. CORLETTE, at the Sydney Hospital.
Afternoon: Demonstrations in the wards and theatres of the Royal Prince Alfred Hospital. Pathological Demonstrations, by DR. KEITH INGLIS, at the Sydney University.
Evening: Demonstrations at the Venereal Diseases Clinic at the Sydney Hospital.

January 17, 1922.

9.30 a.m. to 11 a.m.: Respiratory Diseases, by DR. E. W. FAIRFAX, at the Royal Prince Alfred Hospital.
11 a.m. to 12.30 p.m.: Modern Aspects of Urology, by DR. R. GORDON CRAIG, at the Royal Prince Alfred Hospital.
Afternoon: Demonstrations of Urological Cases at the Royal Prince Alfred Hospital.
Evening: X-Ray Demonstrations of Chest Cases, by DR. H. R. SEAR, at the Royal Prince Alfred Hospital.

January 18, 1922.

Afternoon: Operations, etc., by DR. FOURNESS BARRINGTON, at the Royal Prince Alfred Hospital.

January 19, 1922.

9.30 a.m. to 11 a.m.: Diseases of the Blood, by DR. C. BICKERTON BLACKBURN, at the Royal Prince Alfred Hospital.
11 a.m. to 12.30 p.m.: Ear, Nose and Throat, by DR. H. J. MARKS, at the Royal Prince Alfred Hospital.
Afternoon: Pathological Demonstrations, by PROFESSOR D. A. WELSH, at the Sydney University.

January 20, 1922.

9.30 a.m. to 12.30 p.m.: Demonstrations in Anæsthetics, by DR. M. LIDWILL and DR. S. A. SMITH, at the Royal Prince Alfred Hospital. Infant Feeding, Demonstration and Lecture, by DR. W. F. LITCHFIELD and DR. MARGARET HARPER, at the Royal Prince Alfred Hospital.

The fee for the course is two guineas. Further particulars can be obtained from PROFESSOR FREDERICK A. TODD, Secretary of the Sydney University Extension Board, University of Sydney.

POST-GRADUATE WORK IN MELBOURNE.

THE MELBOURNE PERMANENT COMMITTEE FOR POST-GRADUATE WORK announce that the course held in Melbourne in November was thoroughly successful, 41 practitioners having taken part. The Committee further announce that arrangements will be made for two or more special courses to be held during next winter. Suggestions concerning the subjects for these courses from practitioners will be welcomed. The subjects of selection at present are cystoscopy, renal diseases and problems in obstetric practice.

The Medical Journal of Australia

SATURDAY, DECEMBER 3, 1921.

Tropical Medicine.

IN the discussion on the question of the physiological reaction of the white man to the tropical conditions of Australia at the Australasian Medical Congress, 1920, emphasis was laid on the relative freedom of northern Queensland from so-called tropical diseases. Malaria and filariasis and other affections commonly met with in tropical countries exist in Queensland, but their incidence is said to be low. Rarer forms of tropical diseases are met with in Queensland, the Northern Territory and parts of Western Australia. Apart from these diseases, consideration should be given to those affections which are found in temperate zones, although their characters coincide to some extent with those of the true tropical diseases. Dengue fever, anchylostomiasis, amœbic dysentery and beri-beri are sufficiently common to merit serious attention. In addition to these diseases, which are mildly endemic in certain districts of Australia, there is the still larger problem of exotic diseases which may be introduced into Australia from countries within short sailing distance of our shores. It is held by some that the northern areas of Australia offer no opportunities for the study of tropical diseases. With the single exception of hookworm, no extensive investigation is being carried out in the pathology, ætiology or classification of the diseases commonly present in the north. Attention has been called on several occasions to the fact that it is not yet known which mosquito carries the plasmodium of malaria. Much remains to be discovered in connexion with filariasis, while the problems awaiting solution regarding the infrequent but nevertheless important continued fevers, parasitic infestations and other tropical lesions are many. In the next place Australia has possessions in the north and has the mandatory control over immense tracts of lands close to her own properties. In the Pacific there are innumerable islands, each of which presents a peculiar medical and hygienic proposition. Nowhere in the whole world is there a more fertile field for medical and protozoological research than in the wide Pacific.

The Federal Government has the duty of initiating and maintaining the activities necessary to protect Australia from invasion by disease. The new Department of Health should be the instrument for combating diseases which impair the virility and strength of the community as a whole. The Department is charged with the care of the white and coloured populations in Papua and New Guinea. This represents perhaps the most important function of the Government, for the prosperity and productivity of the people of the Commonwealth depends on their well-being and freedom from devastating disease. It is futile to plead financial stringency as an excuse for a limitation of the policy and programme in regard to health matters. No expenditure is extravagant that is carefully planned and properly devised for the purpose of improving the health of the people. The Government itself tacitly admits this in principle, since it has collaborated with the International Health Board in the Hookworm Campaign and is spending money in a fruitful war on this crippling infection.

The time has arrived when the question should be asked whether it is the intention of the Federal Government to carry out a vigorous and thorough policy in tropical hygiene and medicine. Months have passed since the Institute of Tropical Medicine at Townsville has been allowed to languish without a head and without a *raison d'être*. This excellent establishment is situated centrally for the purpose of the collection of information concerning the tropical diseases of Australia. Its name indicates the nature of the researches that should be carried out. While it is justifiable to utilize this institution for the purpose of the investigation of physiological problems connected with the development of northern Queensland by white men, this matter should be of secondary importance to that of the study of tropical diseases. Care should be taken to prevent the Institute from becoming the playground of politicians with party or personal aspirations. The Institute should be enlarged and extended in order that better and more modern equipment may be installed and wider scope given to the workers. The staff should be enlarged and special departments de-

veloped, to bring it into line with the great research institutions of the world. Lastly, arrangements should be made for members of the staff to have recurring periods of service in laboratories further south for a period of sufficient duration to prevent them from feeling the strain of continuous intense work in a tropical environment. This would cost a considerable sum of money, but if the workers were reasonably salaried and comfortably housed, the expenditure would be justified by the returns.

We learn that the policy of parsimony is being followed in New Guinea. Under the German rule, there were thirteen medical officers working continuously in this vast territory. This number was by no means too large. Colonel A. Honman, the Principal Medical Officer, has been compelled to visit Melbourne to beg for five medical officers to carry out the work of thirteen men. He, no doubt, recognized the futility of demanding a full complement. Australia has a sacred responsibility for the welfare of certainly not less than three-quarters of a million natives. She is satisfied to discharge this duty by appointing five medical officers. Australia has the opportunity of conducting investigations of the most valuable kind in a land teeming with unsolved medical and hygienic problems. She grasps this opporunity by sending a single medical officer to establish a small laboratory in Rabaul. In addition there are the responsibilities and the opportunities centred in Papua. The response is little better here than there.

We are further tempted to ask the Federal Government for a frank statement concerning its policy in regard to the institution of a medical service on the mainland and in the various islands in the Pacific Ocean to probe into Nature's secrets and to add to our knowledge concerning the diseases affecting the native populations and concerning their incidence, ætiology, treatment and prophylaxis. Apart from the humanitarian aspect of this work, there is so much information of first-rate importance to be gained that it is little less than a crime to neglect the opportunity. Work remains to be done within Australia and this form of work can best be conducted as part of a large, organized plan to embrace the lands scattered in the oceans surrounding our continent.

THE AFTER-EFFECTS OF LETHARGIC ENCEPHALITIS IN CHILDREN.

THE name *encephalitis lethargica*, now generally adopted by the profession in all countries, was first suggested by Economo, an Austrian physician, to describe an epidemic disease which appeared in Vienna in the spring of 1917.[1] A year later a similar epidemic made its appearance in England, but as its prevalence corresponded to a period when tinned foods were a common article of diet, it was at first believed that the disease was a manifestation of botulism with associated ocular and other paralyses. The failure to demonstrate the presence of the *Bacillus botulinus* in the infected persons led to the abandonment of this view and the substitution of another—that the disease was an aberrant form of influenza in which a selective attack was made on the central nervous system. This doctrine was promoted by the fact that a marked increase in the incidence of the disease was simultaneous with the outbreak of the appalling epidemic of influenza which swept over Europe and reached Australasia in 1918. The possibility of a relationship between lethargic encephalitis and influenza is certainly supported by several clinical facts, but the biological evidence in favour of their identity is slight. Another suggestion, strongly advocated by F. G. Crookshank in England, that the disease was identical with acute infective poliomyelitis (Heine-Medin) but tended to attack the mid-brain and higher centres rather than the spinal cord, has not been supported with any positive evidence. Acute poliomyelitis commonly attacks children, whereas *encephalitis lethargica* shows no special preference for any age of life. The microscopical hæmorrhages which are a common feature of the former disease, are rare and inconspicuous in the latter. Much speculation has been made in regard to these two diseases and the so-called "X disease" described in Australia by Professor J. B. Cleland and Dr. A. Breinl. The consensus of opinion is that acute poliomyelitis and lethargic encephalitis are distinct disease processes with no relationship to one another. The relationship of Australian "X disease" to either of these conditions is still a matter of dispute.

Such intense interest has been manifested in the ætiology of epidemic encephalitis that the late symptoms and complications of the disease have been largely neglected. Pierre Marie and G. Levy have pointed out that although generalized choreiform movements disappear usually at the end of two or three months, localized rhythmic movements and muscular weakness may persist for a year or more. The tremors and rigidity of the *paralysis agitans* type of the disease may persist for an indefinite period. Little attention, however, has been given to the residual mental symptoms, although the lethargy, irritability, hallucinations and transitory delusions of the disease when at its height have been thoroughly studied.

In a report made to the Local Government Board in 1918 A. S. Macnalty pleaded for caution in con-

[1] *The Quarterly Journal of Medicine*, April, 1921.

nexion with the advancing of any opinion in regard to the after-effects of the disease. He believed it possible that mental changes and residual paralyses might prove to be not uncommon sequelæ. About the same time E. Farquhar Buzzard stated his conviction that epilepsy, mental deficiency, hemiplegia and diplegia would prove to be permanent results of encephalitis occurring in childhood.

Quite recently Drs. Donald Paterson and J. C. Spence have attempted to inquire into the truth of Farquhar Buzzard's prediction.[1] They selected twenty-five children between the ages of three months and eleven years who were without doubt suffering from the disease in a typical form. Three of the four children described by Batten and Still in a paper on "Epidemic Stupor" published in 1918 were included in this series. Batten and Still made no reference to any after-effects exhibited by these patients. Drs. Paterson and Spence found that one of the three was now an inmate of a mental hospital. She showed marked mental deficiency and failed to recognize even her own parents. The second child was dull and slow to learn, lacking the normal child's interest and buoyancy. The third, a boy aged 14 years, who had been a normal, intelligent boy prior to his suffering from *encephalitis lethargica* in 1918, from which he appeared to make a complete recovery, has become a criminal since his illness. Theft is his besetting sin and the police already regard him as an habitual criminal. Of the authors' 25 patients, only one died. Of the remaining 24, only six have completely recovered from their illnesses, no physical or mental trace of which remains. Eighteen show various degrees of mental deficiency. Seven of these show all the mental stigmata of pronounced and permanent idiocy. They are woefully lacking in intelligence, show no recognition of their parents and certainly no affection towards them, drool from the lips and make the quaint grimaces and gestures characteristic of the congenital idiot. The remaining eleven show slighter degrees of mental deficiency—all dating from the illness from which they suffered. Dull and backward, slow to learn, with just sufficient intelligence to avoid danger in the streets, some of them show a moral obliquity which augurs badly for their future. Those whose habits cannot be described as actually criminal, are mischievous and untrustworthy, causing added anxiety to the mother, who finds it difficult enough to control the antics of the average small boy.

But the disastrous effects of the disease are observed in the physical as well as in the mental condition of the children. The children are well nourished, but are slow to develope the characteristics of normal children. Some of them, for example, reach the age of two years before they are able to sit up. The power of standing or walking is likewise delayed. Drs. Paterson and Spence insist that the slow physical development is a result of the mental deficiency and is not due to organic palsies. Seven of the eighteen affected children are still suffering from organic residual paralyses resulting from the disease. Two suffer from typical spastic diplegia, one from hemiplegia with contractures, three from ataxia and incoordination of the arms and one from the so-called Parkinsonian syndrome.

The authors' paper reads like an appalling commentary on the after-effects of the disease. If their main observations are correct (and they certainly appear to have been carefully made), *encephalitis lethargica* has more terrors than the immediate risk of death. Perhaps Dr. Paterson and his colleague happened upon an unusually bad series of children. Further observations and the experiences of other physicians will be anxiously awaited. Nothing concerns a nation more than the welfare of its children.

[1] *The Lancet*, September 3, 1921.

RAT FLEAS AND PLAGUE.

TEN years ago C. J. Martin and Harriette Chick attempted to ascertain the relative affinity of various species of rat fleas for man. In the course of this investigation it was found that the majority of men and women offered small attraction to a rat flea that had a live rat at its disposal. A few individuals proved appetizing to the discriminating *Xenopsylla cheopis*. As would be expected, fleas of all description leave dead rats for living human beings, when other living rats are not available. Dr. R. Newstead and Mr. Alwen M. Evans have investigated the rat flea problem in Liverpool.[1] They distinguished between ship rats, wharf and warehouse rats and city rats. *Mus rattus*, the black rat, was found to be more common than *Mus norvegicus* on ships and docks, while the latter were found in excess in city areas. *Xenopsylla cheopis* infests the black rat. It is the common Indian plague flea. It cannot survive save in the imaginal stages a temperature below 5° C. and consequently it is rarely found in cool climates. This flea was found in relatively large numbers on rats caught on board ship. On the wharves, of 24 black rats, three harboured ten *Xenopsylla cheopis*. Only four fleas of this species were found on warehouse rats. A colony of rats had made their home in close proximity to a steam culvert and 56 specimens of *Xenopsylla cheopis* were counted. Finally, only three fleas of this kind were found in the remainder of the city. *Ceratophyllus fasciatus* is the common rat flea of temperate climates. It was taken in considerable numbers, more frequently with the brown than with the black rat. *Ceratophyllus londiniensis* was found in small numbers. It does not appear to be of importance as a plague vector. *Leptosylla musculi*, on the other hand, is known to be a possible vector of plague bacilli. It is naturally a mouse parasite, but often leaves its usual hosts for rats. These fleas were plentiful, especially in dock warehouses. Only one *Ctenocephalus canis*, the common dog flea, was found, although it is by no means uncommon as a rat parasite. The authors were unable to determine any definite correlation between the frequency of the various species of fleas and atmospheric conditions (temperature, etc.). The only fact of importance in this connexion was the breeding of *Xenopsylla cheopis* close to a hot culvert.

[1] *Annals of Tropical Medicine and Parasitology*, September, 1921.

Abstracts from Current Medical Literature.

THERAPEUTICS.

(240) Aniline Dyes as Urinary Antiseptics.

E. G. DAVIS (*American Journal of the Medical Sciences*, February, 1921) records a study of the antiseptic properties and the renal excretion of 204 aniline dyes. The author postulates that the ideal internal urinary antiseptic should be chemically stable and relatively non-toxic and non-irritating and antiseptic in high dilution in urine (regardless of the reaction of the latter). It should also be eliminated in high percentage by the kidney without injury to the body. Clinically no such drug is known, the drugs in common use as urinary antiseptics falling far short of the ideal. The recognition of the antiseptic properties of certain aniline dyes suggested the possible suitability of drugs of this group as urinary antiseptics. Preliminary investigations of these substances with regard to their antiseptic value on agar plates and in urine and their toxicity and excretion made it possible to eliminate many of the 204 substances as practically unsuitable. Special investigations were made with regard to the powers of the selected dyes to retard the growth of *Bacillus coli* and *Staphylococcus albus*, because of the frequency of infection of the urinary tract by these organisms in the human subject. In accordance with the well-recognized resistance to treatment of *Bacillus coli* infections, it was found that this organism displays *in vitro* great hardiness in spite of the presence of aniline dyes. Only 24 of the 204 dyes studied prevented the growth of the colon bacillus in urine in a dilution of 1 in 1,000. Of these several were effective in alkaline urine only and the majority were antiseptic in higher dilution in alkaline than in acid urine. This fact might prove of clinical importance because the artificial production and maintenance of an alkaline urine is a relatively simple matter. Of the 204 dyes studied, it was found that there were only thirteen which were antiseptic in urine (*in vitro*), which were excreted by the kidney after intravenous injection and which exhibited no toxic properties following moderate dosage (about 20 mgm. per kilogram). It was then attempted to determine whether passage through the blood stream and kidney would interfere with the antiseptic properties and whether sufficient dosage could be safely administered to produce adequate concentration in the urine. From a careful study of the results of his experiments the author was able to select from the 204 dyes studied only fifteen which were antiseptic in urine, excreted by the kidney and relatively non-toxic. With only two of these, proflavine and acriflavine, was it possible to demonstrate the secretion of antiseptic urine following intravenous administration. It was concluded that reasonable expectation existed that a dye suited for use as a urinary antiseptic might be discovered or synthesized. Experiments to date have indicated that dyes of the triphenylmethane, xanthone, acridin and azin groups gave promise of value, but that there was no known drug ideally suited for the purpose of internal urinary antisepsis.

(241) Treatment of Empyema with Gentian Violet.

R. H. MAJOR (*American Journal of the Medical Sciences*, September, 1921) discusses the value of frequent aspiration and of injection of solutions of gentian violet into the pleural cavity in the treatment of post-influenzal empyema. It was found that the policy of delaying operation until a series of preliminary aspirations had been carried out, had the effect of reducing considerably the mortality. In the groups investigated all cases marked by a turbid pleural fluid containing organisms were classed as empyemata, the exudate having varied from a slightly turbid sero-fibrinous exudate to thick creamy pus. Of 312 patients admitted to hospital suffering from influenza, in 40, or 12.5%, empyema developed. Disastrous effects following too hasty operative interference in empyema were uniformly noted during the epidemic of influenza. Preliminary aspiration, therefore, became the treatment of choice in these cases and various forms of closed drainage were employed with or without irrigation with antiseptic solutions. The procedure adopted by the author was as follows: The chest was aspirated by means of a Potain's outfit, the fluid was withdrawn and 100 c.cm. of an aqueous solution of gentian violet were introduced into the pleural cavity through the aspirating needle. The solution was allowed to remain in until the next aspiration. At first a dilution of 1 in 10,000 was used, but this was followed by solutions of increasing strengths till a strength of 1 in 1,000 was reached. In all, 27 patients were treated in this manner, of whom 14, or 51.8%, were cured; 8, or 29.6%, were not cured and were later subjected to operation; and 5, or 18.5%, died. In six of the fourteen successful cases frank pus was obtained at the first aspiration and the fluid continued to be thick and purulent, later becoming thinner and finally disappearing altogether. It appeared that in certain instances, when frank pus was obtained, the disease cleared up after repeated aspiration and the instillation of gentian violet. The average number of instillations necessary varied from 14 to 16. In successful cases two noteworthy features were manifested at an early stage, *viz.*, a gradual lowering of temperature and a diminution of the amount of fluid obtained by thoracocentesis. While admitting that possibly certain of the patients might have recovered without the use of gentian violet instillations, the author considered that the prompt symptomatic response to such injections and the rapid diminution or disappearance of bacteria in the pleural fluid was evidence that the dye-stuff possessed a very definite bactericidal value.

(242) Iodine in Goitre.

S. P. BEEBE (*Medical Record*, June 11, 1921), in a review of the literature and a statement of his own experience of the treatment of goitre by the administration of iodine, states that the iodine content of goitrous glands is less than that of normal thyreoid glands, that the iodine content of goitrous glands is increased by the administration of iodine and that the absorption of iodine frequently assists the reversion to more normal cellular conditions in glands which have become the seat of cellular hyperplasia. Iodine should be given in some form to patients with simple or endemic goitre. Beebe favours the administration of small doses of potassium iodide, 0.06 to 0.12 gramme thrice daily for six weeks, with a gradual increase to at most 0.3 gramme three times a day. Iodide should be administered for three to six weeks at intervals up to two years, according to the improvement made. Iodine is valuable in the treatment of those forms of goitre associated with hyperthyreoidism. The initial dose should be 0.03 to 0.06 gramme of potassium iodide administered thrice daily. Over-dosage will cause increased disturbance. In typical acute Graves's disease in young women iodine should be administered with great caution. The best effects of iodine are seen in women who have had hyperthyreoidism for several years, with goitre, exophthalmos, tachycardia and tremor well marked. Iodine is an essential part of the treatment in every case of hyperthyreoidism, whether the patient is subjected to operation or Röntgen ray therapy or treated by means of rest in bed and general medical measures.

(243) Vaccinotherapy.

JEAN MINET (*La Presse Médicale*, July 13, 1921) reports excellent results obtained by himself in the treatment of acute and chronic pulmonary affections with vaccines in a novel way. In the first place, he used vaccines made from the patient's sputum, a direct smear being examined and the proportions of different organisms present estimated by a count of several fields. A vaccine was then prepared containing the organisms in the same proportion. For example, if the sputum contained staphylococci 70%, streptococci 20% and *Micrococci tetrageni* 10%, the vaccine contained 350,000,000 staphylococci, 100,000,000 streptococci and 50,000,000 *Micrococci tetrageni* in each cubic centimetre. Half to one cubic centimetre of this vaccine was injected every second day for one to ten days. Later, after determining the relative proportions of different bacteria present in the sputum, he used stock vaccines in the same proportions and doses as above. He found that asthma, chronic bronchitis, lobar pneumonia and bronchopneumonia were all very favourably affected by this particular form of treatment.

UROLOGY.

(244) The "Fourth Venereal Disease."

C. W. JEFFERSON (*Urological and Cutaneous Review*, June, 1921) has reviewed the present state of knowledge of the so-called fourth venereal disease. The scientific description of the affection is "specific ulcerative and gangrenous balano-posthitis." The first comprehensive description was given by Bataille and Berdal in 1891. The disorder is now recognized as a distinct entity which may be described as an acute inflammation of the glans and apposed preputial surface, characterized clinically by ulceration which progresses sometimes to gangrene, with an abundant secretion of pus with a putrid odour. The disease is caused by a spirochæte living in symbiotic association with a fusiform bacillus. These organisms are identical with those causing Vincent's angina and noma. The vibrios (fusiform bacilli) are actively motile, Gram-negative and anaerobic. They are best found in the deeper part of the necrotic fissure. The spirochætes are less abundant and are found more superficially. The same spirochæte occurs as a saprophyte in the mouth and is only pathogenic when the above-described fusiform bacilli are also present, when it lives under anaerobic conditions, or when the patient is in a state of greatly lowered resistance. Thus on the penis the spirochæte only causes infection when protected from the air by a long prepuce. The infection of the penis is believed to occur from the saliva. The disease is uncommon in private practice (1 in 200 venereal cases), but in dispensary work the infection is fairly common. The process begins as small excoriations which later coalesce to form superficial ulcers covered with an adherent necrotic pellicle and surrounded by an inflamed border. Preputial œdema and phimosis occur. The superficial form may progress to the gangrenous type, with black sloughing of the parts. Even the whole glans may be shed. The constitutional disturbance is slight, even in the severe gangrenous cases. The period of incubation is about three to five days. The essential part of the treatment is to expose the lesions so that oxygen can reach them. The best application is 2% hydrogen peroxide applied continually as a wet dressing. Circumcision or dorsal incision may be necessary.

(245) Traumatic Factor in Hydronephrosis.

F. C. HERRICK (*Journal of Urology*, January, 1921), discussing the ætiology of hydronephrosis (or pyelectasis, which he suggests as a better name), assigns to trauma an important place. It is now recognized by urologists that the lesser dilatations determinable only by special methods, such as pyelography, are pathological as much as the greater, more obvious pyelectases. The cases in which trauma is a cause, fall into two groups. True hydronephrosis developing within a few days or at the most a few weeks from the injury is very rare. In the second type the patient follows his occupation after a variable period of disability, but later on begins to notice symptoms of pain, frequency and possibly cloudy urine or hæmaturia. The trauma considered here is of the sub-cutaneous, sub-parietal or blunt types, as well as that due to muscular violence. In the first group the relation of the pyelectasis to the trauma is unmistakable, but in the second the history of trauma is easily overlooked and may have occurred one or many years before the consultation. In the traumatic production of pyelectasis the most important factors to be considered are renal mobility and ureteral fixation. A series of blows, falls or repeated muscular strains, when the patient already has a more or less mobile kidney, will aggravate the condition quickly or slowly according to circumstances; this leads to ureteral angulation with consequent back pressure. The problem is analogous to so-called traumatic hernia, an acknowledged rarity, which is the result of congenital factors, trauma being only a contributory cause. In its upper four or five centimetres the ureter leaves its close attachment to peritoneum and passes through fat to the kidney. This upper part of the ureter, which is that part of the duct above the point of crossing of the spermatic vessels, is mobile and follows the movements of the kidney during respiration, strain or the application of violence. Where the vessels cross the duct is the point where angulation is most readily caused by renal mobility, hæmorrhage around the ureter, or scar formation following such hæmorrhage.

(246) Chronic Posterior Urethritis.

E. L. GAUTIER (*Journal d'Urologie Médicale et Chirurgicale*, April, 1921) discusses the diagnosis and treatment of chronic posterior urethritis. The symptoms are often characteristic. A common sign is the occurrence of successive relapses after any excess. Almost always there are sensations of tickling or burning in the region of the posterior urethra. Common genital symptoms that occur are, in order of frequency, frequent nocturnal pollutions, painful ejaculations and blood-stained ejaculations. As regards urinary symptoms, diurnal frequency is one of the most significant. At night the patient may not have to rise or he has to urinate only once. Urgency of micturition indicates extension of the inflammation to the neck of the bladder. Other signs are an increased sensibility of the posterior urethra to instrumentation or a marked intolerance to certain antiseptic solutions, even in low concentration, particularly the oxycyanide of mercury. To study the lesions directly the cysto-urethroscope is unexcelled. The posterior urethra may show lesions while the vesical neck is healthy, but when the bladder neck is affected, the posterior urethra is always implicated as well. The common lesions are simple congestion, œdema leading to soft infiltration, vesicle or papule formation, hard infiltration and polypoid masses. Except in the minor lesions the usual treatment with dilatations and instillations is of little efficacy. Cauterization is indicated, but must not leave dangerous scarring, and for this reason high-frequency destruction is to be preferred, for it can be so applied as to leave supple cicatrices. Sparks should be used and not direct contact. The current should only be passed for a few seconds at a time and the effect observed. With this treatment the immediate and remote results have been excellent.

(247) Female Bladder Symptoms.

E. E. PADGETT (*Urological and Cutaneous Review*, June, 1921) emphasizes the importance of two groups of factors, in addition to sepsis, as causes of vesical distress in women. In one group the cause is pressure from without, resulting in trigonal hyperæmia, a condition which, although predisposing the bladder to infection, is by no means invariably followed by this complication. The majority of patients, however, do not seek advice till the infection has been superimposed on the earlier trouble. Pregnancy excites trigonal hyperæmia by the pressure of the uterus on the bladder. This is especially severe when the gravid organ is retroflexed or incarcerated. The non-gravid uterus may exert pressure on the bladder by its cervix when retroflexed, or by its fundus when antiflexed. Tumours of the uterus or adnexa are likewise a source of pressure on the bladder. The second group includes factors which bring about lack of the normal supports of the bladder. Tears and strains of the vesical supports during labour are the chief causes in this group. The condition of stretching and laceration leads to cystocele, with consequent stagnation and infection of the urine. When the ground is prepared by congestion, organisms may enter by various routes and set up actual cystitis. Perhaps the commonest route is by the short urethra and usually infection by this path follows catheterization. Another route is by a descending renal infection. Primary infection from the blood is rare.

(248) Neoplasms in Undescended Testicles.

J. H. CUNNINGHAM (*Journal of Urology*, May, 1921) declares that a perusal of the literature shows that the development of new growths in undescended testicles is in reality of rare occurrence. He details a case of his own which is the only instance of tumour developing in a testicle which has been replaced in the abdomen from the inguinal canal. A summary of figures from several sources shows that of 452 testicular tumours only 40 developed in organs imperfect in their descent. The prognosis is bad and the mortality high; the majority of patients die within one year after the operation and in one series it was noted that only 3 out of 59 patients were alive two years after operation.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Western Australian Branch was held at the Perth Hospital on July 20, 1921, DR. G. W. BARBER, C.B., C.M.G., D.S.O., the President, in the chair.

Hereditary Optic Atrophy.

DR. C. MORLET, O.B.E., read a paper on hereditary optic atrophy as a menace to the community. A genealogical table was exhibited and two members of the affected family were presented for examination. (See page 499.)

A discussion ensued.

DR. J. E. F. STEWART moved that the information concerning this family should be sent to the Public Health Department.

The motion was seconded by DR. J. J. HOLLAND.

DR. D. D. PATON held that there was no danger to the community from this condition. It did not consider it necessary to notify the health authority. He moved an amendment to the effect that no notification be sent to the Public Health Department until the transmission of diseases and deformities had been recognized as public health matters.

The amendment was seconded by DR. O. P. PAGET.

DR. JUETT expressed the opinion that the public health authorities should be notified for record purposes.

The amendment was put to the meeting and was lost. The motion was carried.

Congenital Deficiency of the Œsophagus.

DR. W. J. BEVERIDGE read a short note on congenital deficiency of the œsophagus. He exhibited a pathological specimen. A similar specimen derived from a patient who had been in the Children's Hospital in 1913 was also shown.

Dental Neuralgia.

DR. F. GILL presented a patient with dental neuralgia. The pain had been very severe. The nerve had been divided at its entrance to the inferior dental foramen on the left side of the jaw.

Foreign Bodies.

DR. H. BALDWIN GILL exhibited two foreign bodies. The first had been removed with the aid of bronchoscopy, while the second had been removed with the aid of œsophagoscopy.

A MEETING of the New South Wales Branch was held on September 9, 1921, in the B.M.A. Building, 30-34, Elizabeth Street, Sydney, DR. FOURNESS BARRINGTON, the President, in the chair.

DR. A. A. PALMER and DR. STRATFORD SHELDON recorded a large number of interesting experiences from the Coroner's Court and demonstrated *post mortem* and other exhibits in connexion with recent medico-legal cases.

Cut-Throat.

DR. A. A. PALMER first introduced an unusual case of cut-throat. He pointed out that, as a rule, the evidence which they had to give in cases of alleged suicide or alleged murder, was evidence of fact. Occasionally opinions had to be expressed in regard to whether a wound was self-inflicted or not. Both Dr. Sheldon and he had given a very large amount of consideration to this particular case and had arrived at a definite conclusion after repeated revision of the facts.

They had been asked to examine a woman whose throat had been cut. The wound measured nearly 27 cm. in length and extended from a point 1.25 cm. below and rather more than 2.5 cm. posterior to the inferior attachment of the left pinna to a point on the right side nearly corresponding to that on the left. The line of the incision was a curved one, its centre being 3.8 cm. from the point of the chin. It passed through the thyreo-hyoid membrane immediately below the hyoid bone. The left great cornu of the hyoid bone was cut through near its extremity, as was the posterior wall of the pharynx. The wound was deeper in the centre and on the right side than on the left. On the left side of the neck the main vessels had apparently not been cut, but a large tributary of the internal jugular vein had been severed close to the main vessel. On the right side the carotid artery had been severed and the median and anterior walls of the internal jugular vein had been removed to the vertical extent of 2.5 cm. to 3 cm.. The *longus colli* muscles and the anterior longitudinal ligament contained five cuts. Four of these were very distinct and one at least had penetrated the whole thickness of the prevertebral tissues down to the bone. The two longest were about 2.5 cm. in length and the others about 1.25 cm.. The distance from the highest cut to the lowest was 2.5 cm..

The wound as it passed from the left side to the right became gradually deeper. At the right extremity the skin was undercut, *i.e.*, the incision in the deeper tissues extended further than the incision in the skin.

Dr. Sheldon and he had arrived at the opinion that the wound was homicidal. The reasons given were: (*i.*) The great size and extent of the wound, (*ii.*) the cutting of the front of the vertebral column in so many places, (*iii.*) the undercutting at one extremity of the wound, (*iv.*) the situation and direction of the wound. It was very rare for a suicidal wound to commence as high up as was the case in this woman. If a suicidal wound commenced at this level, it would almost certainly have terminated at a lower level. In this case the wound turned up again, to end at a similarly high level. (*v.*) The deceased was a right-handed woman. She would probably have cut from left to right. Usually a suicidal wound was deepest at or near its commencement and then tailed or tapered off. This wound became deeper toward the right side. Dr. Palmer thought that it was probable that the wound had been made from left to right on account of the undercutting at the right end. Moreover, in front of the vertebral column the tissues were cut upwards toward the skull for some distance. The edges of the wound were jagged; from the lower edge another more superficial wound extended downwards and to the right. This wound began 3.8 cm. to the right of the middle line and measured 5.7 cm. in length. In his opinion the wound did not exhibit a single characteristic of a suicidal wound. He mentioned that Dr. Sydney Jamieson had made an examination of the specimen without having knowledge of the particulars of the case and in a full report had agreed with their finding in every particular. The specimen was exhibited.

Cardiac Death.

In the next place Dr. Palmer related the details of a case in which the *post mortem* evidence had been insufficient to enable them to determine the real cause of death. A married woman, aged 24 years, had suddenly collapsed and died after walking hurriedly up a slope. It was stated she had been warned that it would be unsafe for her to have an anæsthetic during her confinements, owing to the condition of her heart. *Post mortem* it was found that the heart weighed 185 grammes. No definite disease was detected. A long, thin, apparently fibrous band, resembling a *chorda tendinea*, crossed the cavity of the ventricle. No changes were discovered on microscopical examination. The kidneys were slightly enlarged and there was some pitting of the surface after the capsule had been removed. The convolutions of the brain were somewhat flattened, as though from cerebral hæmorrhage. No hæmorrhage was found, but merely an increased quantity of cerebro-spinal fluid.

The Falleni Case.

Dr. Palmer procedeed to give a very full account of the medico-legal aspect of the celebrated Falleni case. On October 3, 1917, Dr. Palmer and Dr. Sheldon had made a *post mortem* examination of a woman found dead in the scrub at Lane Cove. They had been told that an empty whisky flask and a bottle smelling of kerosene had been found close to the body. The features were unrecognizable. The greater part of the body was extensively charred, but the legs and feet had escaped. The ribs on the left side were burnt through, as well as part of the anterior abdominal wall. The nose, mouth, chest, abdomen and vagina had been attacked by maggots. All the internal organs bore evidence of having been cooked. The stomach con-

tained a considerable quantity of food, some of which resembled chicken.

The endometrium was thickened and the cavity of the uterus contained a small amount of blood clot. The arms and hands were much contracted.

The stockings on the legs were not burnt. The skin of the legs was smooth and elastic. There was considerable blistering in the neighbourhood of the left knee and in the front aspect of the right leg. The blisters contained thick fluid. Their floors were red and they were surrounded by red areolæ.

In the absence of other evidence, Dr. Palmer and Dr. Sheldon had assumed that death had probably been due to burning. The Coroner returned an open verdict.

On July 22, 1920, the body was exhumed and the coffin was opened in the presence of Drs. Palmer and Sheldon. As the police had suggested the possibility of bullet wounds, Dr. J. G. Edwards was asked to make a number of skiagrams. No bullet was located. Much of the soft parts of the body still remained as a kind of adipocere which was either separated or could be easily detached from the bones in cakes. The skull, from which the calvarium had been removed at the previous examination, was practically bare. In the thin bones surrounding the air sinuses and other cavities there were many cracks. These were best seen in the sphenoid and in both maxillæ. They had obviously been caused by the heat of the fire. There was, however, another crack or linear fracture the cause of which was not so certain. This was situated in the right parietal bone and extended backwards almost parallel to and about 3.8 cm. above the squamous suture; it ended posteriorly in the right limb of the lambdoid suture by dividing into two. The fracture measured 5.7 cm. in length and involved the whole thickness of the bone for about 4.5 cm.. Below this the lambdoid and the occipito-mastoid sutures appeared to be opened up and at one point the inner plate projected inward. From the occipito-mastoid suture just behind the jugular process of the occipital bone a linear fracture ran backwards and inwards to the *foramen magnum*.

Dr. Palmer and Dr. Sheldon had formed the opinion that this fracture had probably been caused by violence and not by the heat of burning. Others who had seen it, found no reason to doubt that it was caused in the same way as the other fractures.

Learned counsel for the defence had asked Dr. Palmer whether it was not a fact that a bone, such as the parietal, consisted of 50% of water. Dr. Palmer repied that he did not think that it was as high as 50%. Counsel quoted Halliburton, who had stated that it was 50%. Dr. Palmer expressed his surprise and stated that he was not convinced.

Later on Dr. Palmer had looked up the matter in the literature. The majority of authorities gave the percentage of water in bone at about 50%. Noel Paton, however, had found it to be 10%. At. Dr. Palmer's request, Mr. W. N. Doherty, the second Government Analyst, carried out an analysis of bone taken from fresh bodies with proper precautions. The soft tissues were removed hastily and the bones wrapped in water-proof paper and handed to the analyst. The result of the analysis was that the moisture contained in various bones was found to be relatively low. The percentages were given as follows:

Parietal bone of a male aged 57 years ..	10.06%
Parietal bone of a female aged 31 years ..	8.67%
Femur with marrow of a male aged 50 years—	
Middle third	13.42%
Other portion	14.00%
Femur of full-grown fœtus	11.73%
Femur without marrow (shaft) of male aged 45 years	12.52%
Lower epiphyseal end of the femur of adult male	11.93%
Rib of adult male	38.18%

Dr. Palmer proceeded to read a record of the circumstances in connexion with the arrest and conviction of Eugène Falleni, *alias* Harry Crawford, on a charge of murder. This person was the daughter of an Italian residing in New Zealand. There were eleven children in the family. None except the woman in question had given any trouble. She had been born in Florence in 1875 and had arrived in New Zealand with her parents when two years of age. As she had grown up, she had been in the habit of running away from home dressed in boy's clothing. At times she did not return for many days. It had been learnt that during her absence from home she had worked as a boy at various brickyards and laundries. She was not given to immorality at this stage. While in her teens she had run away from home with the skipper of an Italian ship, whom she accompanied to various countries. On returning to Newcastle she had parted from the skipper after a quarrel. She had then been engaged as cabin boy on board a sailing ship in charge of an Italian skipper. For some time prior to this she had been wearing boy's clothes. A year or so later she had been brought back to Newcastle by the skipper with an infant girl which had been born at sea. Eugène Falleni had then adopted the name of Henry Crawford and was employed at an hotel in Sydney as a "useful." During the following years she had worked at various hotels, laundries and factories. When she was arrested, she was employed at an hotel at Annandale.

Some years before Falleni had met a widow with a boy aged nine years. The two had become very friendly and eventually Falleni, under the name of Crawford, had induced the widow to leave her employment and to start a small business. Crawford had lived with the widow until February, 1919, when they had married. The marriage had been an unhappy one, largely on account of Crawford's drinking habits and filthy language. Crawford had separated from the widow for a time, but later had rejoined her. The daughter of Falleni, *alias* Crawford, had been cast on the world, but had returned to her mother shortly before giving birth to an illegitimate child. At this stage the daughter had referred to Crawford as her father. Some neighbours had endeavoured to help her to locate her mother. On one occasion they had plied the daughter with numerous questions concerning the whereabouts of her mother, until the girl, in desperation, had said :"I can't keep it any longer. That is mother sitting there dressed in man's clothes." The fact had been hushed up for a time, but eventually reached the ears of the widow. The widow had taxed Crawford with the charge and, as a result, had compelled her to undress, which she did, disclosing that she was a woman. After further consideration it was agreed between the two that the widow should secure a judicial separation, to prevent any unpleasant disclosures which might influence the future welfare of her son. The relations of the two naturally had become strained and Falleni, although continuing to work, began to drink very heavily. On September 28, 1917, Falleni and the widow had been seen leaving their home. One of them was carrying a cane suit case. The widow was not again seen alive. On the same day Crawford (Falleni) had been seen in the vicinity of the paper mills at Lane Cove. On the following day Crawford had again been seen in the same neighbourhood. The widow's son had been staying away from home and had returned on October 1, 1917. Crawford was drinking whisky and had asked the boy to participate, but he had refused. He had inquired for his mother and had been informed that she had gone to North Sydney with some friends. On October 2, 1917, the charred remains of a woman had been found at the spot where Crawford and the widow had been seen on September 28. The police had been notified, investigations followed and the Coroner returned an open verdict.

On October 3, 1917, Falleni sold the household effects of the home in which she and the widow had lived. It was stated that Falleni, *alias* Crawford, had performed acts of cruelty on the son of the widow and had endeavoured to do away with him in a well-known place on the cliffs near Sydney.

Nearly three years later the boy of the widow had related many circumstances connected with Crawford to his aunt and had told her that he had come for the purpose of endeavouring to ascertain where his mother was. The statements of the boy were related to the police. Further investigations were carried out and an endeavour was made to identify the exhibits connected with the Coroner's inquiry into the cause of death of the woman whose charred remains had been found near the paper mill at Lane Cove. The boy and his aunt had identified several articles as having been in the possession of the widow. The upper and lower sets of false teeth found with the body were recognized by a den-

tist as having been made for this woman. It was also discovered that Falleni had sold a diamond ring belonging to the woman.

Falleni was traced to an hotel in Annandale and was arrested. She stated that her name was Harry Crawford, that she was a Scotsman, born in Edinburgh.

Dr. Palmer had been instructed to examine the prisoner, in order that marks of identification could be noted. At first she had refused to allow herself to be examined. On learning that treatment in gaol in the first instance involved bathing in the presence of warders and changing of clothing, Falleni asked whether she could be placed in the women's ward. This request was refused and she then confided to the detectives the fact that she was a woman. Dr. Palmer confirmed the truth of the statement after an examination.

In a signed statement given before this disclosure Falleni maintained that she was a man. The statement contained the alleged history of her movements and included a reference to a marriage which had been contracted at a registry office in September, 1919. The detectives therefore confronted Falleni with this woman, who was known as Mrs. Harry Crawford. During the search Falleni had told the detectives that there was an article in a leather trunk which she did not wish "Mrs. Harry Crawford" to see. This article was an artificial penis which Falleni had used in her various sexual adventures. The article was shown at the meeting. It was a large dummy organ made of firm material and covered with gauze, while an outer skin covering protected about five-sixths of its length. It was quite straight. Dr. Palmer explained that when found that portion uncovered by tissue had been much soiled. When "Mrs. Harry Crawford" was told that Falleni was a woman, she had refused to believe it. She had stated that no one could have had a better opportunity of knowing the sex of Falleni and further had claimed that she was three months pregnant "to the laddie." It appeared that this woman had reached the climacteric. At a later date this woman had informed the police that Falleni had told her that "he" was a widower with one daughter; that "he" had married for a second time a widow with a son and that this son had caused considerable trouble. Falleni was a very heavy smoker and drinker. The detectives discovered that Falleni was in the habit of visiting two married women in a suburb of Sydney during the absence of their husbands. Each of these women had become aware that Falleni visited the other. One of them, actuated by ill-feeling and jealousy, had made a hole in the weatherboard wall of the other woman's bedroom and, looking through this hole, had witnessed cohabitation between Falleni and the second woman.

Falleni had been tried at the Central Criminal Court in Sydney, had been found guilty and had been sentenced to death. The sentence had subsequently been commuted to penal servitude.

Post-Mortem Examinations.

DR. STRATFORD SHELDON started his demonstration with the exhibition of a table setting forth the work of the Coroner's Court from 1914 to 1920. He pointed out that *post mortem* examinations were not ordered in every case. He proposed to speak of some of their omissions and errors. Unless there were a distinct indication to look for a certain lesion, it was very easy to miss it. Sometimes when *rigor mortis* was present, great difficulty might be experienced in the examination of the mouth. In one case they had learned after the examination had been completed that the tongue and lower jaw had been removed surgically some time before for malignant disease. In the same way it was quite possible to miss poisoning in an alleged drowning case.

Dr. Sheldon made a diversion by telling a story to illustrate the humorous side of Dr. Palmer's character. On one occasion Dr. Palmer had asked him to look after the mortuary work on Christmas Day and he had agreed. Early on Christmas morning he was surprised to receive a telephone message from Dr. Palmer to the effect that he was down at the mortuary and that there was an unusual amount of work to do. Dr. Sheldon went off post haste and found Dr. Palmer in the dead house surrounded by the usual unattractive, unsavoury and depressing material. Dr. Palmer looked up from his morbid occupation, held out his hand and said: "A merry Christmas!"

Dr. Sheldon explained the usual procedure in making *post mortem* examinations for the Coroner. The police report, which was compiled from various statements, had to be received with caution and an open mind. Immediately after each examination the gloves and instruments were sterilized ready for the next examination. This was not done for the benefit of the subject. Contrary to the general impression, everything was not revealed at the examination. The history of the manner of death was often most important. He quoted several cases in which no opinion could have been offered had there been no history.

Cerebral Hæmorrhage.

Dr. Sheldon dealt with the subject of cerebral hæmorrhage. He drew attention to six cases in quite young women. In three of them the hæmorrhage was in the frontal lobe. He was able to give an explanation of the cause in only one. There was a remarkable case of pontine hæmorrhage in a primipara, aged 22 years, who, two hours after confinement was eating her breakfast, complained of pain in the head and died almost at once. The previous history of this woman was in every way satisfactory. Details of other cases were also given. Before leaving this subject he pleaded for more general surgical interference in extra-dural hæmorrhage the result of a ruptured middle meningeal artery with or without fracture of the skull.

Œdema of the Lungs.

In the next place he drew attention to the common occurrence of acute œdema of the lungs. He contrasted this condition with the acute hæmorrhagic œdema of influenza with pulmonary signs and also with the acute frothy œdema of drowning. In drowning the lungs were usually voluminous and a fine froth appeared at the nose and mouth when the chest was squeezed. A few days after drowning the fluid escaped from the lungs into the pleural cavity. After about a week the lungs resembled the lungs of a new-born infant. Dr. Litchfield had expressed the opinion that the fluid came from inside and not from outside in these cases. He, Dr. Sheldon, agreed with this view, but thought that some fluid might come from outside in some cases. He spoke of several cases of drowning in which the lungs were found to be dry. Two of these were in young people who had bathed with distended stomachs.

He related the instance of a young, healthy Chinaman who had eaten just over 90 grammes of rice and who, on carrying a box upstairs, had fallen dead. A man, aged 79 years, had gorged just under one kilogram of a mixed diet in a restaurant and promptly died.

Unusual Causes of Death.

The death of two other Chinamen was noted. One of them, a man aged 31, had died of a subdiaphragmatic abscess which had burst into his pericardium. This abscess was the result of infection from a liver fluke, *Distomum sinense*. Hundreds of these liver flukes had been found in his bile passages. The other Chinaman had been caught by four men while visiting a lady. They had inserted a small salad oil bottle through the anus into his rectum. The Chinaman had taken a three-cornered file bent to a hook and had attempted to deliver the bottle with it. The attempts had been unsuccessful, but the hook had penetrated the wall of the bowel. He had been admitted to hospital, where the bottle had been expressed by pressure from above. The man had died of peritonitis three days later. A man had visited a lady in a Sydney suburb and had died in her house at an extremely critical moment. It was found that the death had been caused by the rupture of an aneurysm.

He also referred to the Tuck case. This individual was said to have been a sexual pervert and had been killed by one of his victims. Dr. Sheldon had accompanied the police for the purpose of inspecting the premises and had found in Tuck's apartment pieces of cotton wool, vaseline pots, powder pots, towels and flagellators like wire fly swats. A diary was found in which the writer had described his sadistic sensations.

Death by Choking.

The next class of case dealt with was choking. A man in a sailors' home had put a piece of meat measuring 12.5 cm. by 5 cm. into his mouth. The meat had stopped at his glottis and had choked him. A girl, aged 18 years, had been admitted to the Coast Hospital on account of diphtheria. A month later she had been discharged after bacteriological examination had failed to reveal any diphtheria bacilli in the throat. Two days after her return home she had started to choke at a meal and had died immediately. The *post mortem* examination had revealed a cast of the trachea which had been dislodged against her glottis.

In another case a child had found her mother strung up to a bed post. The police discovered false teeth, as well as some of the deceased's teeth, under the bed. Death had been caused by choking. One of the jurors at the trial was a builder by trade. The husband of the deceased was also a builder. This juror had noted that the woman had been tied up by a rein and that the knot employed to secure it was a builder's knot.

A young man had hanged himself in a curious fashion. He had placed some books on a table in front of a mirror of a wardrobe and, placing a bar from the end of the bed to these books, he passed a window cord over the bar, tying one end to his scrotum and the other with a loop round his neck. He was stark naked and hanged himself in this way.

Bullet Wound of the Skull.

Dr. Sheldon described the means adopted to account for a curious course of a bullet in a celebrated case. The wife of a policeman had been shot in the head. The skull showed both a wound of entry and a wound of exit of the bullet. Dr. Sheldon pointed out that the usual effect was for the bullet to penetrate the skull and to be held up against the inner side of the opposite wall. Tests had been carried out on sheep with bullets purchased from Mick Simmons, Limited, and with the authorized police bullets. Only one of the former, but all but one of the latter, had gone right through both sides of the skull.

Criminal Abortion.

The next subject dealt with was criminal abortion. Dr. Sheldon stated that the effect of interference with the uterus was very varied. In many cases the infection travelled behind the peritoneum, in others through the broad ligament leading to purulent peritonitis, in others again there was a rapid infection of the blood without any local signs and the patients died very rapidly. The different effects, he thought, were due to different organisms. In the case of those who died very rapidly, decomposition had often been noticed which had obviously begun before death. He gave an account of a number of cases of air embolism. Many pathologists attributed this condition to gas-forming organisms. In some of his cases such an explanation could not be maintained. He quoted ten cases during the past few yearse. He spoke of an instance in which death had occurred in the surgery of an irregular practitioner immediately after the uterus had been irrigated by means of a Higginson's syringe which had contained air. He contrasted with this case that of a woman who had died in the sixth month of pregnancy. The examination had been carried out a few hours after death and it had been found that the blood and amniotic fluid had been infected with *Bacillus aerogenes capsulatus*.

He spoke of two women who had been treated for criminal abortion by drainage of Douglas's pouch. Both were suffering from acute miliary tuberculosis. Neither was pregnant; the one had previously been pregnant, while the other had not.

In the last place, Dr. Sheldon spoke of a case of complete transposition of the organs. In the course of his remarks he had demonstrated a number of specimens.

Death from Drowning.

DR. W. F. LITCHFIELD remarked that the demonstrations of Drs. Palmer and Sheldon had been most valuable and instructive. He wished to say a few words concerning the condition of the lungs after death from asphyxia. He thought that the signs could be systematized. In the first place, œdematous or wet lungs arose under very definite conditions. If death occurred from asphyxia, as in cerebral hæmorrhage, the pulmonary œdema resembled that associated with drowning. In the next place he recognized the condition of dry lungs such as occurred after death from fibrinous bronchitis. In this condition there was no œdema. Similarly, in laryngeal diphtheria, when there was extension of membrane from the larynx downwards into the lung, there was no œdema. Taylor and other authorities had stated that the lungs after death from asphyxia were sometimes wet and sometimes dry. Dr. Litchfield held the view that when œdema of the lungs was present, the left side of the heart failed first; when the lungs were dry the right side of the heart gave out before the left. In fibrinous bronchitis there was excessive inspiratory effort; blood was drawn into the thorax, the heart became distended and paralysis and embarrassment of the right side of the heart resulted.

After death from drowning the water found in the lungs was not fluid that had been inspired. It was a true œdema. He assumed that the effect of the cold water impinging on the larynx was to liberate an expiratory effort in the form of coughing. The stomach of a person who had been drowned, was always full of water. Water in the trachea was often regurgitated from the stomach before or soon after death. Dr. Litchfield claimed that this explanation satisfied all the conditions associated with drowning.

Discussion.

THE HONOURABLE J. B. NASH, M.L.C., congratulated the speakers and referred in appreciatory terms to their frankness in admitting their inability to ascertain the cause of death in every case. He testified to the thoroughness with which Dr. Palmer carried out his duties.

DR. RALPH WORRALL pointed out that the Falleni case illustrated in the most admirable manner the extreme innocence of many women and some men in regard to the sexual function. He held that in women the development of the maternal instinct sometimes lessened the sexual instinct. Referring to one of the cases mentioned by Dr. Sheldon in which the existence of an acute miliary tuberculosis had been missed, he stated that all the evidence in the history of the patient pointed to an abortion. He had seen several cases of air embolism. Some of these patients recovered when the amount of gas absorbed into the circulation was small. After delivery in the left lateral position the patient should be at once turned on to her back to prevent the aspiration of air into the vagina and uterus. If this were not done and forcible compression of the uterus were made, the air in the uterus might easily be forced into the open blood vessels.

DR. JOHN MACPHERSON related a case of pontine hæmorrhage in a lodge patient. The diagnosis had been very difficult and the practitioner, who had made a lucky guess at the diagnosis, had scored heavily over the doctor immediately concerned. Dr. MacPherson also dealt with the case of a man who had been brought to the hospital unconscious. He had died without regaining consciousness. At the *post mortem* examination it was found that the gall bladder was adherent to the transverse colon and that the bowel was completely obstructed by an enormous gall stone, which had passed through a fistulous opening between the two viscera. At times the history given by the patient's friends was too explicit. Dr. MacPherson related the case of a child alleged to have been bitten by a snake. When he arrived at the house, he had found the father sucking the buttock. The father had informed him that the child had gone to bed, that a snake had been curled up in the bed and that it had bitten the boy on the buttock. Dr. MacPherson immediately cut out the affected area and applied permanganate of potash. There were no symptoms of snake venom poisoning. He then examined the bed to see whether the snake was still present. All he found was a pin sticking through the sheet slightly blood stained.

DR. ARCHIE ASPINALL said that the story of the Chinaman and the salad oil bottle reminded him of a story told him by another practitioner of a patient who had deliberately inserted a small castor oil bottle into the rectum to produce pressure on the prostate and to prevent semen from passing during coitus. He had also seen a patient in hospital who had inserted a vaseline pot through the anus to control

the hæmorrhage from piles. The jar had slipped into the rectum and had been removed with difficulty.

DR. H. S. STACY congratulated both Dr. Palmer and Dr. Sheldon on their interesting communications and on their cheerful optimism. He made some remarks on cases of septic meningitis following fractured skull. The meningitis had manifested itself several months after the trauma. He also spoke of the extensive laceration of brain substance as a result of a gun-shot wound, the mark of which was scarcely visible in the scalp. He thought that many cases labelled shell shock or neurasthenia were in part due to an organic lesion. He pleaded for more frequent *post mortem* examinations. They were not carried out even in police cases, unless they were necessary for the purpose of distinguishing between natural and violent death. By *post mortem* examinations they were able to learn of their mistakes.

DR. OLIVER LATHAM referred to a case of cerebral hæmorrhage associated with a large cerebral tumour which Dr. Froude Flashman, Dr. John Wallace and he had described. At that time it had been pointed out that many cases of cerebral hæmorrhage were due to glioma. For instance, Powers had maintained that apoplexies with optic neuritis were probably gliomatous. Recently Dr. Prior had remitted a small piece of cerebral tissue from a mental patient, in which some yellowish discolouration happened to attract his attention. Section revealed a definite glioma of the large celled type, involving an important area of the cortex.

DR. C. E. CORLETTE referred to the case of a woman who had been admitted to hospital with the diagnosis of cerebral hæmorrhage. She was hemiplegic and aphasic and was treated with calomel. Following this there was a swelling of the abdomen and the patient vomited. She died and at the *post mortem* examination a large, recent hæmorrhage was found. It was discovered, however, that death had been due to an internal hernia. A loop of bowel had slipped under by a band stretching from the broad ligament to the region of the appendix and had become strangulated there.

DR. FOURNESS BARRINGTON thanked Dr. Palmer and Dr. Sheldon for their very instructive and important demonstrations. He also wished to direct the attention of the members to some of the specimens lying on the table. They were the work of an artist. These specimens had been prepared by Dr. Keith Inglis. Turning to the question of criminal abortion, he pointed out that interference was not uncommon when the patient was not pregnant. He had found the end of a bone crochet hook in Douglas's pouch and in the abdomen and other instruments used for the purpose elsewhere. These were examples of the amenorrhœa of fear. He thought sometimes that the interests of science had to be subserved and cited details of a case in which the difficulties of obtaining a valuable pathological specimen had been overcome by thoughtful co-operation.

At a very late hour DR. PALMER rose to reply. He pointed out that he had not been able to discover the pathological basis in cases of cerebral hæmorrhage in young people. In regard to snake-bite, he recalled an instance of a man who had been playing with a snake without fangs. It appeared that the venom sac was still present. The man had died. In conclusion, Dr. Palmer stated that he had never seen a murderer on trial who was not quite calm. Usually they appeared innocent.

DR. STRATFORD SHELDON replied very briefly.

THE WAR MEMORIAL FUND IN VICTORIA.

THREE additional names have been added to the list of subscribers to the War Memorial Fund of the Victorian Branch of the British Medical Association. They are: James S. Buchanan, Allan Robertson, H. Hume Turnbull.

The sub-committee of the Victorian Branch dealing with the Memorial, after exhaustive inquiries, has compiled the following list of medical practitioners of Victoria who gave their lives in the service of the country during the great war. The convenor, DR. J. W. DUNBAR HOOPER, of 2, Collins Street, Melbourne, invites readers to forward to him the names of any other Victorian practitioner who fell during the war.

G. Grantham Anderson, F. S. Bond, N. J. Bullen, S. J. Campbell, E. W. Deane, A. F. Deravin, G. S. Elliott, J. F. Fairley, A. R. Fox, Shenton Garnet, R. H. Gibbs, H. F. Green, E. E. Harkness, A. V. Honman, G. Howitt, W. W. Hearne, M. R. Hughes, Johnson Hughston, D. D. Jamieson, F. Miller Johnson, Eric Kerr, A. W. H. Langley, Keith M. Levi, C. R. Lister, J. G. Mackenzie, G. C. Mathison, G. P. Merz, A. Guy Miller, J. J. Nicholas, C. J. Oliver, W. Rogerson, A. C. Rothera, P. B. Sewell, Harold South, C. Alwyn Stewart, Harold A. Teague, E. R. Welch, M. L. Williams, A. H. O'Hara Wood, Leonard A. Wright.

Public Health.

THE PLAGUE OUTBREAK.

THE following information is culled from the Bulletin of the Commonwealth Department of Health, Nos. 17 and 18, of November 22 and 25, 1921:

Plague in Man.

On November 19, 1921, the total number of cases of plague affecting human beings was 71. There had been 38 deaths. The distribution is given as follows: Townsville, 26 cases with 16 deaths; Brisbane, 25 cases and one suspected case with 13 deaths; Cairns, 14 cases with eight deaths; Port Douglas, two cases with one death; Toowoomba and Mackay, one case each; and Innisfail, one suspected case.

On November 18 a child, aged 15 years, was removed from South Brisbane to the Wattlebrae Isolation Hospital on account of plague. On the following day a labourer, aged 23 years, who had been employed by the Queensland Preserves, Limited, in South Brisbane, was also admitted. The boy died on November 20 and the labourer died on November 24.

On November 22 a further suspected case was reported from the Milton district. On November 24 a yardsman, aged 40 years, was admitted from an hotel in Wickham Street, Brisbane.

There were two fresh infections in human beings at Townsville. The first was in a young man who was isolated on November 17 and died on the following day. The second patient was a wharf labourer, aged 64, who had been loading sugar on November 16 and 17. The disease commenced on November 17 and the patient died on November 21. It was held that the source of infection was not from the vessel.

Two further cases in human beings were reported from Cairns, the first on November 21 and the second on November 23.

Plague in Rats and Cats.

During the week ended November 19, 1921, ten infected rats out of a total of 1,703 examined were discovered in Brisbane. Eleven cats were examined during the week and one taken from a draper's store in South Brisbane was found to be infected.

During the following days up to November 24, 1921, only two infected rats were taken in Brisbane.

At Townsville five infected rats were discovered during the week ended November 19. Six further infected rats were reported on November 21.

At Cairns a large number of rats was caught and examined, but none was infected. The officer in charge of the Commonwealth Plague Laboratory reported that 65 fleas from rat carcases were examined. Of these, 64 were *Xenopsylla cheopis* and one was *Ctenopsylla musculi*.

In the Hinchinbrook district two rats were found dead. Smears from these rats revealed *Bacillus pestis*. Eighty-nine other rats were free from infection. From Innisfail smears of eighteen rats were received at the laboratory during the week ended November 19. Of these, four proved to be infected.

The examination of rats taken at Thursday Island, in Sydney, in Tasmania and in Western Australia failed to reveal any plague-infected animals.

The *Nardoo* arrived at Melbourne from Lucinda Point, *viâ* Townsville, on November 12, 1921, with a cargo of sugar. Three rats were caught during unloading and one of these was found to harbour organisms resembling *Bacillus pestis*. The vessel was completely fumigated and was subjected to the usual procedure, pending the result of further bacteriological investigation.

Correspondence.

WARMED ETHER ANÆSTHESIA.

SIR: In an article of mine on the heat losses connected with ether anæsthesia published in THE MEDICAL JOURNAL OF AUSTRALIA of August 6 and August 13 last, I demonstrated certain facts, based on exact physical measurements, which were not in accord with certain preconceived theories that have been expounded on the subject of warmed ether anæsthesia. These theories had never before been scrutinized in this way. It seems that the theories had been erected into gods and had received a good deal of worship. People do not like having their gods scrutinized like common things—weighed, measured, judged and perhaps rejected as false. Dr. Dyring has been disturbed over my article and has brought forward a "Rejoinder" in your issue of to-day's date. It is written in most courteous terms and no one could possibly take exception to the mode of his attack. I would now, in advance, ask Dr. Dyring to forgive me if in my reply I hit hard. For though I am sure he is an accomplished anæsthetist, he is weak on the physiological side and I shall have to prove it.

Dr. Dyring seems to think that a method of etherization which he and others find satisfactory, must stand condemned if the facts are accepted. Let me say that he is entirely mistaken. It is false theory that has to be given up. I myself said of the warmed ether practice, but not of the theory supplied by its advocates: "Looking at it as a whole, I am sure it may well rank as a most valuable improvement in the art of etherization." Dr. Dyring quotes this, but describes it as a "respectable euthanasia." Well, why may I not be credited with honestly meaning what I said? I meant it. If Dr. Dyring and other anæsthetists find empirically that they get good results by any of their methods, who questions their results? But it is quite another matter if they want us to shut our eyes, open our mouths and swallow their explanatory theories, theories which have been weighed in the balance, tested with scruplous care and found wanting.

Dr. Dyring "craves permission to examine the subject shorn of its mathematical embroideries." Embroideries! If we are to get at the real facts, we must use measurements and leave vague impressions alone. I placed the following aphorism of Lord Kelvin at the head of my article: "When you can measure what you are thinking about and express it in numbers, you know something about it; but when you cannot measure it, when you cannot express it in numbers, your knowledge is of a meagre and unsatisfactory kind; it may be the beginning of knowledge, but you have scarcely in your thoughts advanced to the stage of science." This does not please everybody. For instance, my critic, who rejects this plan and craves permission in a question of physics to examine it, not in the way by which, according to Lord Kelvin, "you will know something about it,' but by which he would go back to what is "meagre and unsatisfactory." The permission he craves cannot be given. Numerical statements are not "embroideries"; they are absolutely fundamental to clear and accurate thinking—just what we want in these matters, if we want the truth. And my critic winds up by advising me that I am not competent to examine the subjects concerned. Well, I ought to be. And I cannot admit his suggestion, however courteously made, that it is outside my province.

Now let us look at the substitute for scientific methods offered to us (through Dr. Dyring) by two ponderous pseudo-scientists, Messrs. Gwathmey and Davis. Thus the former :"In 26 patients anæsthetized with warmed ether vapour there was a loss of body temperature averaging 0.29° F., as against the loss of 1.02° F. in 40 cases anæsthetized under similar conditions with the open drop method." Note the "under similar conditions." So they were all similar excepting for the method of anæsthetization. That is a "large order" by itself. It means, amongst many other things, that their heat production was similar. Was it? And why speak of "loss" of temperature. Temperature rises or falls. We can lose organic heat, which is a form of energy, but not temperature. To speak of loss of temperature does not conduce to clear thinking. I alluded to this tendency in my article when I remarked that "it had become obvious that anæsthetists had been thinking about it (the warmed ether subject) in terms of temperature when they ought to have thought in terms of heat." What puerile futility to imagine that a comparison of the body-temperature before and after an anæsthetic expresses the heat loss! The level of the temperature depends on the balance between heat production and heat loss. If heat production does not keep pace with heat loss, the temperature must fall. If these temperature observations give Dr. Gwathmey trustworthy information about the quantity of heat loss, why not use it to tell us what was the amount of heat production. If it tells about one, it surely will tell us about the other. Perhaps that is how he found that the heat production was similar in all the cases. It seems that the measurement of metabolism has now been shorn of all difficulties. Or is it that Dr. Gwathmey is making it quite evident that he has not even a *Kindergarten* acquaintance with the subject of metabolism and that his "science" is mere charlatanism. Dr. Dyring has allowed himself to be bewitched by ridiculous piffle like this.

Dr. Gwathmey's calibre may be judged from another of Dr. Dyring's quotations: "It is interesting to note that even a fraction of a degree of elevation of temperature of the anæsthetic agent above that of the room will have a marked effect in maintaining the body temperature of the patient." I take it that Dr. Dyring will not attempt to question my proof that the quantity of heat absorbed by raising the temperature of the vapour of 100 c.cm. of ether from 0° C. to 33° C. is 0.903 of a large calorie. Dr. Gwathmey tells us (and Dr. Dyring swallows it) that a fraction of a degree (F. °, not C. °) will have a marked effect. A rise of one degree F. (not a fraction of one degree) in the temperature of the vapour of 100 c.cm. of ether means a difference in cooling value of 15 small calories, or $^1/_{60}$ of one large calorie, and the heat loss it would counteract would be that represented by the evaporation of less than half a drop of sweat. The calculation can be worked out by Dr. Dyring by mental arithmetic in a few seconds. This quantity of heat is to have a "marked effect" on the body temperature of a patient who is losing anything from 150 to 250 large calories (150,000 to 250,000 small calories), an hour by other means. This implies absolutely the colossal limit of credulity. Cannot Dr. Dyring see that he has been too unsuspicious and has been hoaxed?

Now we come to Dr. Dyring's own particular mare's nest, the ace of trumps that he uses as the cap and finisher of everything. The problem is the measurement of heat loss involved by the respiration of one cubic metre (1,000 litres) of air, measured as expired air at 33° C. and at standard barometric pressure. Let us suppose that we have constructed a satisfactory apparatus and proceed to receive the expired air into a calibrated gasometer, so that when an indicator reaches a certain mark we have collected exactly 1,000 litres. When we have collected it, the gasometer contains exactly 1,096.7 grammes of dry air and exactly 35.3 grammes of water-vapour, no matter what the temperature and hygrometric condition of the inspired air. Let us take the case of a man inspiring air at 0° C. and saturated for that temperature, since absolutely dry air is never breathed. At the end of the experiment, the gasometer will contain 1,096.7 grammes of dry air, *plus* 4.4 grammes of water-vapour inhaled with the atmospheric air, *plus* another 30.9 grammes of water-vapour. The total water-vapour present, as above stated, must be 35.3 grammes. Where did that other 30.9 grammes of water-vapour come from? The only possible source was the body of the patient. And where did the latent heat of vaporization for that 30.9 grammes come from? Again, the only possible source was the body-heat of the patient. Contrary to Dr. Dyring's belief, which is founded on a misconception, it does not matter one scrap whether the patient rebreathed the expired air in the mask, or rebreathed backwards and forwards into the gasometer itself. Rebreathing merely makes a tidal reflux during the process of accumulation, but until the indicator shows 1,000 litres, the 1,096.7 grammes of dry air have not been collected and when that is reached, it is impossible not to have the 35.3 grammes of water-vapour also. The whole thing is absolutely simple and the calculation of heat loss in calories is then within the capacity of any schoolboy. Yours, etc.,

Sydney, November 19, 1921. C. E. CORLETTE.

Naval and Military.

APPOINTMENTS.

THE following information appeared in the *Commonwealth of Australia Gazette*, No. 91, of November 24, 1921:

Australian Military Forces.

The Governor-General in Council has approved of the following appointments being made in the Australian Military Forces, with effect from 1st September, 1921, unless otherwise stated:

STAFF.

Army Head-quarters.—LIEUTENANT-COLONEL T. E. V. HURLEY, C.M.G., is seconded from the Australian Army Medical Corps, Third Military District and is appointed Assistant Director of Medical Services; CAPTAIN M. J. HOLMES, D.S.O., is seconded from the Australian Army Medical Corps, Third Military District, and is appointed Director of Hygiene and is granted the temporary rank of Major whilst holding such appointment.

FIRST MILITARY DISTRICT.

Eleventh Mixed Brigade.—COLONEL A. G. BUTLER, D.S.O., Australian Army Medical Corps, is appointed Assistant Director of Medical Services (Fifth Divisional Head-quarters).

SECOND MILITARY DISTRICT.

First Cavalry Division.—MAJOR R. W. W. WALSH, D.S.O., Australian Army Medical Corps, is appointed Deputy Assistant Director of Medical Services, Divisional Head-quarters.

First Division.—MAJOR W. VICKERS, D.S.O., Australian Army Medical Corps, is appointed Deputy Assistant Director of Medical Services, Divisional Head-quarters.

Second Division.—CAPTAIN AND BREVET-MAJOR G. C. WILLCOCKS, O.B.E., M.C., Australian Army Medical Corps, is appointed Deputy Assistant Director of Medical Services, Divisional Head-quarters, and is granted the temporary rank of Major whilst holding such appointment.

District Base.—CAPTAIN H. SUTTON, Australian Army Medical Corps, is appointed Assistant Director of Hygiene and is granted the temporary rank of Major whilst holding such appointment.

Australian Army Medical Corps.—COLONEL J. A. DICK, C.M.G., V.D., is appointed to command the Ninth Field Ambulance, with pay and allowances of Lieutenant-Colonel; LIEUTENANT-COLONEL W. C. GREY is appointed to command the Sixteenth Field Ambulance; LIEUTENANT-COLONEL E. S. STOKES is appointed to command the Eighth Field Ambulance; LIEUTENANT-COLONEL J. C. STOREY, O.B.E., is appointed to command the Fourteenth Field Ambulance; LIEUTENANT-COLONEL (provisionally) R. DICK is appointed to command the Second Cavalry Field Ambulance; LIEUTENANT-COLONEL A. L. DAWSON, D.S.O., is appointed to command the Fourth Cavalry Field Ambulance; LIEUTENANT-COLONEL (provisionally) J. J. HOLLYWOOD is appointed to command the First Field Ambulance; LIEUTENANT-COLONEL (provisionally) R. A. P. WAUGH is appointed to command the Fifth Field Ambulance.

THIRD MILITARY DISTRICT.

Second Cavalry Division.—MAJOR H. J. WILLIAMS, D.S.O., Australian Army Medical Corps, is appointed Deputy Assistant Director of Medical Services, Divisional Head-quarters.

Third Division.—MAJOR J. H. ANDERSON, C.M.G., C.B.E., Australian Army Medical Corps, is appointed Deputy Assistant Director of Medical Services, Divisional Head-quarters.

Fourth Division.—MAJOR R. W. CHAMBERS, D.S.O., Australian Army Medical Corps, is appointed Deputy Assistant Director of Medical Services, Divisional Head-quarters.

District Base.—MAJOR S. W. PATTERSON, Australian Army Medical Corps, is appointed Assistant Director of Hygiene.

Australian Army Medical Corps.—LIEUTENANT-COLONEL J. A. H. SHERWIN is appointed to command the Fifteenth Field Ambulance; LIEUTENANT-COLONEL (provisionally) J. J. MCMAHON is appointed to command the Tenth Field Ambulance; LIEUTENANT-COLONEL (provisionally) N. L. SPIERS is appointed to command the Fourth Field Ambulance; LIEUTENANT-COLONEL B. QUICK, D.S.O., is appointed to command the Second Field Ambulance; LIEUTENANT-COLONEL (provisionally) W. W. W. CHAPLIN is appointed to command the Fifth Cavalry Field Ambulance; LIEUTENANT-COLONEL (provisionally) F. C. BURKE-GAFFNEY is appointed to command the Sixth Field Ambulance; LIEUTENANT-COLONEL E. R. WHITE is appointed to command the Third Cavalry Field Ambulance.

FIFTH MILITARY DISTRICT.

Thirteenth Mixed Brigade.—MAJOR C. MORLET, D.S.O., Australian Army Medical Corps, is appointed Deputy Assistant Director of Medical Services, temporarily (Fifth Divisional Head-quarters).

Australian Army Medical Corps.—LIEUTENANT-COLONEL R. S. MCGREGOR, D.S.O., is appointed to command the Thirteenth Field Ambulance.

TRANSFERS TO NEW ORGANIZATION.

The Governor-General in Council has approved of the undermentioned officers serving, on the 30th March, 1921, with units of the Old Organization, the Unattached List, the Reserve of Officers, etc., being transferred, appointed or promoted to the following units under nomenclature of the New Organization, with the ranks stated against their respective names and with regimental or corps seniority in the order shown, such transfers, appointments, promotions, etc., to take effect from the 31st March, 1921, unless otherwise stated:

SECOND MILITARY DISTRICT.

Army Medical Corps.

Australian Army Medical Corps.—COLONEL R. J. MILLARD, C.M.G., C.B.E., Australian Army Medical Corps, and to be supernumerary to the establishment of Colonels, with pay and allowances of Lieutenant-Colonel, from 31st March, 1921, to 31st July, 1921, inclusive; COLONEL J. A. DICK, C.M.G., V.D., Australian Army Medical Corps, and to be supernumerary to the establishment of Colonels, with pay and allowances of Lieutenant-Colonel; COLONEL K. SMITH, C.M.G., Australian Army Medical Corps, 1st August, 1921; COLONEL F. A. MAGUIRE, D.S.O., Australian Army Medical Corps, and to be supernumerary to the establishment of Colonels, with pay and allowances of Lieutenant-Colonel, from 31st March, 1921, to 31st July, 1921, inclusive; LIEUTENANT-COLONELS W. C. GREY, E. S. STOKES, J. H. PHIPPS, D.S.O., A. H. TEBBUTT, D.S.O., and J. C. STOREY, O.B.E., Australian Army Medical Corps; MAJOR R. DICK, Australian Army Medical Corps, 31st March, 1921, and to be Lieutenant-Colonel (provisionally), 1st September, 1921; LIEUTENANT-COLONEL A. L. DAWSON, D.S.O., from the Reserve of Officers, 1st September, 1921; MAJORS J. J. HOLLYWOOD and A. J. MACKENZIE, Australian Army Medical Corps, 31st March, 1921, and to be Lieutenant-Colonels (provisionally), 1st September, 1921; MAJOR P. FIASCHI, O.B.E., Australian Army Medical Corps, 31st March, 1921, and to be Lieutenant-Colonel, 1st September,1921; MAJOR R. A. P. WAUGH, Australian Army Medical Corps, 31st March, 1921, and to be Lieutenant-Colonel (provisionally), 1st September, 1921; MAJORS R. A. ROBERTSON, L. W. DUNLOP, C. H. E. LAWES, W. L. KIRKWOOD, O.B.E., E. M. RAMSDEN, W. T. NEWTON and A. P. WALL, MAJOR (HONORARY LIEUTENANT-COLONEL) C. E. WASSELL, D.S.O., MAJOR W. K. INGLIS, MAJORS (HONORARY LIEUTENANT-COLONELS) C. W. THOMPSON, D.S.O., M.C., and H. V. P. CONRICK, D.S.O., MAJORS A. C. ARNOLD, G. E. MAROLLI and F. L. BIGNELL, D.S.O., Australian Army Medical Corps; CAPTAIN R. W. W. WALSH, D.S.O., CAPTAIN (HONORARY MAJOR) W. H. DONALD, CAPTAIN A. L. BUCHANAN, CAPTAINS (HONORARY MAJORS) W. VICKERS, D.S.O., and T. M. FURBER, CAPTAINS B. C. KENNEDY and C. L. CHAPMAN, D.S.O., CAPTAINS (HONORARY MAJORS) G. C. BYRNE, D.S.O., L. COWLISHAW and C. L. S. MACINTOSH, D.S.O., and CAPTAIN (HONORARY LIEUTENANT-COLONEL) L. W. BOND, D.S.O., Australian Army Medical Corps, 31st March, 1921, and to be Majors, 1st September, 1921; CAPTAIN (HONORARY MAJOR) F. MCINTYRE, M.C., CAPTAIN J. A. JAMES, CAPTAIN AND BREVET-MAJOR J. B. F. MCKENZIE, CAPTAINS W. F. MATTHEWS, W. R. C. BEESTON, J. MCPHERSON and L. MAY, D.S.O., M.C., CAPTAINS (HONORARY MAJORS) W. EVANS, M.C., and R. B. NORTH and CAPTAIN H. H. WILLIS, Australian Army Medical Corps; CAPTAIN (provisionally and temporarily) E. H. KNOWLES, Australian Army Medical Corps, and to be Captain (provisionally); CAPTAIN (HONORARY MAJOR) J. M. ALCORN, CAPTAIN AND BREVET-MAJOR G. C. WILLCOCKS, O.B.E., M.C., CAPTAINS (HONORARY MAJORS) E. A. SANBROOK and N. M. A.

ALEXANDER, CAPTAINS J. C. DOUGLAS and W. W. MARTIN, Australian Army Medical Corps; CAPTAIN (provisionally and temporarily) P. A. L. QUESSY, Australian Army Medical Corps, and to be Captain (provisionally); CAPTAIN C. W. SINCLAIR, CAPTAIN (HONORARY MAJOR) E. H. RUTLEDGE, CAPTAINS R. G. WOODS, C. ANDERSON, M.C., H. SUTTON, N. E. B. KIRKWOOD, M.C., T. F. BROWN, D.S.O., A. M. DAVIDSON, O.B.E., P. A. C. DAVENPORT, J. W. FARRAR, O.B.E., H. G. LEAHY, M.C., E. W. B. WOODS, M.C., and E. E. PITTMAN, Australian Army Medical Corps; CAPTAIN (provisionally) D. T. SMITH, Australian Army Medical Corps; CAPTAINS C. F. ROBINSON, M.C., R. J. HUNTER, R. J. SILVERTON, E. K. PARRY, M.C., A. J. COLLINS, D.S.O., M.C., and K. S. PARKER, M.C., Australian Army Medical Corps; CAPTAIN A. M. ASPINALL and CAPTAIN (provisionally) F. R. CUMMING, Australian Army Medical Corps, 1st May, 1921; CAPTAIN L. T. ALLSOP, M.C., Australian Army Medical Corps, 16th May, 1921; CAPTAIN (provisionally) T. E. PARKER, Australian Army Medical Corps, and to be Captain, 16th May, 1921; CAPTAIN T. W. VAN EPPEN and CAPTAIN (provisionally) R. V. GRAHAM, Australian Army Medical Corps, 1st June, 1921; MAJOR A. J. MOLLISON, from the Reserve of Officers, and to be Captain, 20th June, 1921; CAPTAIN W. K. W. FLOOK, from the Reserve of Officers, 22nd July, 1921; LIEUTENANT (provisionally) O. E. J. MURPHY, Australian Army Medical Corps, 31st March, 1921, and to be Captain (provisionally), 1st September, 1921; LIEUTENANTS (provisionally) A. J. METCALFE, C. R. SMITH, C. B. LEVICK, D. B. LOUDON and W. J. CHAPMAN, Australian Army Medical Corps; LIEUTENANT L. J. SCOTT, Australian Army Medical Corps; LIEUTENANT (provisionally) F. A. E. LAWES, Australian Army Medical Corps, 1st May, 1921; LIEUTENANT (provisionally) E. ROSANOVE, Australian Army Medical Corps, 31st May, 1921; LIEUTENANTS (provisionally) H. W. T. CHENHALL, M. B. FRASER and N. E. GOLDSWORTHY, Australian Amry Medical Corps, 1st June, 1921; GAVIN BRUCE WHITE, to be Lieutenant (provisionally), 16th July, 1921; JOHN IRVINE HUNTER, to be Lieutenant (provisionally), 1st September, 1921.

THIRD MILITARY DISTRICT.

Army Medical Corps.

Australian Army Medical Corps.—COLONEL R. M. DOWNES, C.M.G., Australian Army Medical Corps, and to be supernumerary to the establishment of Colonels, with pay and allowances of Lieutenant-Colonel, from 31st March, 1921, to 6th July, 1921, inclusive; COLONEL R. FOWLER, O.B.E., Australian Army Medical Corps, and to be supernumerary to the establishment of Colonels, with pay and allowances of Major, from 31st March, 1921, to 31st July, 1921, inclusive; COLONEL W. E. SUMMONS, O.B.E., Australian Army Medical Corps, and to be supernumerary to the establishment of Colonels, with pay and allowances of Lieutenant-Colonel, from 31st March, 1921, to 31st July, 1921, inclusive; LIEUTENANT-COLONELS G. G. SHAW, D.S.O., and J. A. H. SHERWIN, Australian Army Medical Corps; MAJORS J. J. MCMAHON and N. L. SPIERS, Australian Army Medical Corps, 31st March, 1921, and to be Lieutenant-Colonels (provisionally), 1st September, 1921; MAJORS (HONORARY LIEUTENANT-COLONELS) G. A. W. J. KNIGHT, O.B.E., and J. J. BLACK, D.S.O., MAJOR T. E. V. HURLEY, C.M.G., and MAJOR (HONORARY LIEUTENANT-COLONEL) B. QUICK, D.S.O., Australian Army Medical Corps, 31st March, 1921, and to be Lieutenant-Colonels, 1st September, 1921; MAJORS W. W. CHAPLIN, F. C. BURKE-GAFFNEY and H. R. CATFORD, Australian Army Medical Corps, 31st March, 1921, and to be Lieutenant-Colonels (provisionally), 1st September, 1921; CAPTAIN (HONORARY LIEUTENANT-COLONEL) E. R. WHITE, Australian Army Medical Corps, 31st March, 1921, and to be Lieutenant-Colonel, 1st September, 1921; CAPTAIN R. A. R. WALLACE, Australian Army Medical Corps, 31st March, 1921, and to be Major (provisionally), 1st September, 1921; CAPTAIN (HONORARY MAJOR) R. L. ROSENFIELD, CAPTAIN AND BREVET-MAJOR (HONORARY LIEUTENANT-COLONEL) A. M. WILSON, D.S.O., CAPTAIN R. W. CHAMBERS, D.S.O., CAPTAIN (HONORARY LIEUTENANT-COLONEL) B. M. SUTHERLAND, O.B.E., CAPTAIN H. J. WILLIAMS, D.S.O., CAPTAIN (HONORARY LIEUTENANT-COLONEL) D. D. CADE, D.S.O., CAPTAIN J. K. ADEY, O.B.E., and CAPTAIN AND BREVET-MAJOR J. H. ANDERSON, C.M.G., C.B.E., Australian Army Medical Corps, 31st March, 1921, and to be Majors, 1st September, 1921; CAPTAINS S. G. L. CATCHLOVE and F. E. KEANE, Australian Army Medical Corps, 31st March, 1921, and to be Majors (provisionally), 1st September, 1921; CAPTAIN N. H. FAIRLEY, O.B.E., Australian Army Medical Corps, 31st March, 1921, and to be Major, 1st September, 1921; CAPTAIN C. C. MARSHALL, Australian Army Medical Corps, 31st March, 1921, and to be Major (provisionally), 1st September, 1921; CAPTAIN E. CHAMPION, CAPTAINS (HONORARY MAJORS) D. C. PIGDON, J. K. RICHARDS and R. S. WHITFORD, CAPTAIN (HONORARY LIEUTENANT-COLONEL) E. W. GUTTERIDGE and CAPTAINS J. V. GRIFFITH and W. B. CRAIG, D.S.O., Australian Army Medical Corps, 31st March, 1921, and to be Majors, 1st September, 1921; MAJOR S. W. PATTERSON, from the Reserve of Officers, 1st September, 1921; CAPTAINS (provisionally and temporarily) S. G. STRAHAN and M. JACOBS, Australian Army Medical Corps, and to be Captains (provisionally); CAPTAINS A. W. NANKERVIS, A. V. R. HANSEN, J. I. CONNOR and G. E. COLE, D.S.O., CAPTAIN (provisionally) W. E. HARRISON, CAPTAINS J. C. P. STRACHAN and G. A. BIRNIE, M.C., CAPTAIN (provisionally) C. W. ADEY, CAPTAIN H. B. LEE, D.S.O., M.C., CAPTAIN (provisionally) J. A. TROUP and CAPTAIN S. CRAWCOUR, Australian Army Medical Corps; CAPTAINS F. J. B. MILLER and F. W. FAY, M.C., and CAPTAIN (provisionally) F. W. TATE, Australian Army Medical Corps, 1st April, 1921; CAPTAINS I. BLAUBAUM, R. C. WITHINGTON, A. P. DERHAM, M.C., and H. A. S. NEWTON and CAPTAIN (provisionally) F. W. JACKSON, Australian Army Medical Corps, 1st May, 1921; CAPTAINS H. G. LOUGHRAN and M. B. O'SULLIVAN, Australian Army Medical Corps, 1st June, 1921; CAPTAIN M. J. HOLMES, D.S.O., HONORARY MAJOR M. W. CAVE, D.S.O., MAJOR W. W. S. JOHNSTON, D.S.O., M.C., HONORARY CAPTAIN H. B. GRAHAM, D.S.O., M.C., CAPTAINS J. C. M. HARPER, M.C., K. C. PURNELL, M.C. J. W. YOUNG and T. S. CAMPBELL, HONORARY CAPTAIN T. G. S. LEARY and CAPTAIN H. C. DISHER, from the Reserve of Officers and to be Captains, 1st July, 1921; ALBERT WILLIAM SHUGG and HAROLD VICTOR CANTOR, to be Captains (provisionally), 31st August, 1921; LIEUTENANT (provisionally) J. H. D'AMER-DREW, LIEUTENANT G. H. BRANDIS and LIEUTENANT (provisionally) W. J. P. FLANAGAN, Australian Army Medical Corps, 31st March, 1921, and to be Captains (provisionally), 1st September, 1921; HAROLD ISAAC ROBINSON to be Captain (provisionally), 1st September, 1921; LIEUTENANT (provisionally) B. H. QUIN and D. L. YOFFA, Australian Army Medical Corps; LIEUTENANTS (provisionally) A. E. LEE, K. D. FAIRLEY and H. E. PEARCE, Australian Army Medical Corps, 1st April, 1921; LIEUTENANT (provisionally) R. SOUTHBY, Australian Army Medical Corps, 1st June, 1921; CHARLES EVANDER VASS SUTHERLAND, CHARLES JAMES OFFICER BROWN, ERNEST WESLEY CHENOWETH, CHARLES HARWOOD OSBORN, GEORGE EDWIN FOREMAN, EDWARD HOLBROOK DERRICK, CHARLES HUGH HEMBROW, JOHN HORACE KELLY, ERIC ECCLES MACKAY, HENRY NEWMAN MORTENSEN and RUSSELL EXON BUTCHART to be Lieutenants (provisionally), 1st September, 1921; LOUIS VICTOR DARBY and OSWALD ROBERT TRUMPY to be Lieutenants (provisionally), 14th September 1921; MAJOR AND QUARTERMASTER O. J. LAWSON, Australian Army Medical Corps.

FIFTH MILITARY DISTRICT.

Army Medical Corps.

Australian Army Medical Corps.—COLONEL G. W. BARBER, C.B., C.M.G., D.S.O., V.D., Australian Army Medical Corps, and to be supernumerary to the establishment of Colonels, with pay and allowances as for Lieutenant-Colonel, from 31st March, 1921, to 31st July, 1921, inclusive; LIEUTENANT-COLONEL R. S. MCGREGOR, D.S.O., Australian Army Medical Corps; MAJOR J. P. KENNY, Australian Army Medical Corps; CAPTAIN C. H. SHEARMAN, Australian Army Medical Corps, 31st March, 1921, and to be Major, 1st September, 1921; MAJOR C. MORLET, D.S.O., from the Reserve of Officers, 1st September, 1921; JOHN DALE, O.B.E., to be Major, 2nd September, 1921; CAPTAIN (temporarily) E. C. EAST, Australian Army Medical Corps, and to be Captain; CAPTAINS W. J. BEVERIDGE and D. I. R. SMITH, Australian Army Medical Corps; CAPTAINS J. BENTLEY, M.C., T. R. JAGGER, M.C., R. C. BASSETT, A. JUETT and H. B. GILL, Australian Army Medical Corps, 1st July, 1921; MAJOR M. B. JOHNSON, from the Reserve of Officers and to be Captain, 1st September, 1921; LIEUTENANTS (provisionally) A. W. FARMER, R. H. MORGAN and W. A. COLLOPY, Australian Army Medical Corps, and to be supernumerary to the establishment, 1st June, 1921.

Books Received.

A MANUAL OF DISEASES OF THE STOMACH, by William MacLennan, M.B., with the assistance of J. Salisbury Craig, M.B., Ch.B.; 1921. London: Edward Arnold & Company; Demy 8vo., pp. 392, with four plates and 31 illustrations in the text. Price: 21s. net.

MIND AND ITS DISORDERS: A TEXT-BOOK FOR STUDENTS AND PRACTITIONERS OF MEDICINE, by W. H. B. Stoddart, M.D., F.R.C.P.; Fourth Edition, 1921. London: H. K. Lewis & Company, Limited; Demy 8vo., pp. 592, with 84 illustrations, including ten plates. Price: 22s. 6d. net.

LECTURES ON THE SURGERY OF THE STOMACH AND DUODENUM, by James Sherren, C.B.E., F.R.C.S.; 1921. London: H. K. Lewis & Company, Limited; Crown 8vo., pp. 96. Price: 4s. 6d. net.

PERMEATING MASTOID MENINGITIS: A CLINICAL NOTE FOR THE GENERAL PRACTITIONER, by J. B. Pike, M.R.C.S. (Eng.), L.R.C.P. (Edin.); 1921. Bristol: John Wright & Sons, Limited; London: Simpkin, Marshall, Hamilton, Kent & Company, Limited; Crown 8vo., pp. 24. Price: 1s. net.

Corrigendum.

A TEXT-BOOK OF GYNECOLOGY, by James Young.—A. & C. Black, Limited, inform us that the price of this book is 15s. net and not 20s., as published in the issue of November 26, 1921.

Medical Appointments.

DR. T. S. GREENAWAY (B.M.A.) has been appointed Medical Officer of the Venereal Clinics and Dispensaries in Brisbane and Medical Officer of the Venereal Isolation Hospital at South Brisbane, Queensland.

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DR. J. MAYE (B.M.A.) has been appointed by the Government of Queensland a Health Officer at Gladstone for the purposes of *The Health Acts, 1900 to 1917*.

* * * *

DR. C. G. GODFREY (B.M.A.) has been appointed Acting Inspector of Inebriate Institutions in Victoria from November 11, 1921, during the absence of DR. W. E. JONES.

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DR. R. W. LETHBRIDGE (B.M.A.) has been appointed Acting Inspector-General of the Insane, Victoria, from November 11, 1921, during the absence of DR. W. E. JONES.

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DR. N. B. CHARLTON has been appointed a Quarantine Officer in the Federal Service.

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DR. H. G. WALLACE, Adelaide, DR. J. H. S. JACKSON (B.M.A.), Brisbane, DR. N. B. WATCH, Perth, DR. T. C. BACKHOUSE, Adelaide, and DR. E. W. H. SMALPAGE, Sydney, have been appointed Medical Officers in the Federal Service for New Guinea at a salary of £1,000 *per annum* without the right of private practice.

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DR. J. KENNY (B.M.A.) has been appointed Honorary Medical Officer (temporary), Public Hospital, Fremantle, Western Australia.

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DR. R. W. MILLER has been appointed Resident Medical Officer, Kalgoorlie Hospital, Western Australia.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xx.

BENEVOLENT SOCIETY OF NEW SOUTH WALES: Junior Resident Medical Officer of Royal Hospital for Women, Paddington.

BROKEN HILL AND DISTRICT HOSPITAL: One Senior and Two Junior Resident Medical Officers.

THE MEDICAL JOURNAL OF AUSTRALIA: Assistant Editor.

ROYAL NORTH SHORE HOSITAL OF SYDNEY: Honorary Assistant Physician for Diseases of the Skin (Fresh Applications).

SYDNEY HOSPITAL: Honorary Relieving Assistant Physician.

WALTER AND ELIZA HALL INSTITUTE OF RESEARCH IN PATHOLOGY AND MEDICINE, MELBOURNE: Temporary Qualified Assistant.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C..

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney	Australian Natives' Association Ashfield and District Friendly Societies' Dispensary Balmain United Friendly Societies' Dispensary Friendly Society Lodges at Casino Leichhardt and Petersham Dispensary Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney Marrickville United Friendly Societies' Dispensary North Sydney United Friendly Societies People's Prudential Benefit Society Phœnix Mutual Provident Society
VICTORIA: Honorary Secretary, Medical Society Hall, East Melbourne	All Institutes or Medical Dispensaries Australian Prudential Association Proprietary, Limited Manchester Unity Independent Order of Oddfellows Mutual National Provident Club National Provident Association
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane	Brisbane United Friendly Society Institute Stannary Hills Hospital
SOUTH AUSTRALIA: Honorary Secretary, 3, North Terrace, Adelaide	Contract Practice Appointments at Renmark Contract Practice Appointments in South Australia
WESTERN AUSTRALIA: Honorary Secretary, 6, Bank of New South Wales Chambers, St. George's Terrace, Perth	All Contract Practice Appointments in Western Australia
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington	Friendly Society Lodges, Wellington, New Zealand

Diary for the Month.

DEC. 6.—Victorian Branch, B.M.A.: Ballot Papers for Election of Office-Bearers returned.
DEC. 6.—New South Wales Branch, B.M.A.: Ethics Committee.
DEC. 7.—Victorian Branch, B.M.A.: Annual Meeting.
DEC. 8.—Brisbane Hospital Clinical Society.
DEC. 9.—Queensland Branch, B.M.A.: Annual Meeting.
DEC. 9.—New South Wales Branch, B.M.A..
DEC. 9.—Queensland Branch, B.M.A.: Council.
DEC. 9.—South Australian Branch, B.M.A.: Council.
DEC. 13.—Tasmanian Branch, B.M.A.: Meeting.
DEC. 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
DEC. 14.—Melbourne Pædiatric Society (Victoria).
DEC. 15.—Victorian Branch, B.M.A.: Council.
DEC. 20.—New South Wales Branch, B.M.A.: Medical Politics Committee; Organization and Science Committee.
DEC. 23.—Queensland Branch, B.M.A.: Council.
JAN. 3.—New South Wales Branch, B.M.A.: Council (Quarterly).
JAN. 10.—New South Wales Branch, B.M.A.: Ethics Committee.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, B.M.A. Building, 30-34, Elizabeth Street, Sydney. (Telephone: B. 4635.)